# A PROSPECTIVE STUDY OF ROLE OF MINIMALLY INVASIVE APPROACHES FOR RENAL SALVAGIBILITY IN MANAGEMENT OF EMPHYSEMATOUS PYELONEPHRITIS

*Dissertation submitted in partial fulfillment of the requirements of*

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***BRANCH 1V – UROLOGY***

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## CERTIFICATE

This is to certify that this dissertation entitled **"A Prospective Study of Role of minimally invasive approaches for renal salvagibility in management of Emphysematous Pyelonephritis"** is the bonafide work done by **Dr. S. Saraswathi**, under our direct guidance and supervision in the Department of Urology, Kilpauk Medical College Hospital & Govt. Royapettah hospital, Chennai,, in fulfilment of regulations of the Tamil Nadu Dr. M.G.R. Medical University for the award **of M.Ch Higher Speciality degree, Branch IV – Urology** during this period of study from December 2012 – November 2014.

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## ABBREVIATIONS

| | | |
|---|---|---|
| DM | - | Diabetes mellitus |
| UTI | - | Urinary tract infection |
| EPN | - | Emphysematous pyelonephritis |
| E.coli | - | Escherichia coli |
| K.pneumoniae | - | Klebsiella pneumoniae |
| B.fragilis | - | Bacteroides fragilis |
| ATP | - | Adenosine tri phosphate |
| NAD | - | Nicotinamide adenine dinucleotide |
| USG | - | Ultrasonogram |
| CT scan | - | Computerised Tomographic scan |
| IVU | - | Intravenous urogram |
| SD | - | Standard deviation |
| TC | - | Total count |
| Hb | - | Haemoglobin |
| PCD | - | Percutaneous drainage |
| PCN | - | Percutaneous nephrostomy |

# INTRODUCTION

# INTRODUCTION

Emphysematous pyelonephritis was first coined by Schultz and Klorfein [2] and is applied when gas is formed and collected only in or around the kidney[3.] Emphysematous pyelonephritis was reported earlier by Kelly and MacCallum in 1898[1] and was considered to be rare. It has got multiple terminologies, such as renal emphysema, pneumonephritis, pyelonephritis, emphysematosa and pneumonephrogram. As pointed by Schultz and Klorfein, emphysematous pyelonephritis is the preferred designation[4].

Necrotizing lesions in infected tissue in diabetic patients or those with an obstructive urinary tract infection by Gas-forming bacteria which uses glucose as a substrate is EPN. Complication of acute sepsis results in a poor prognosis. This disease creats an urologic emergency[5]. Mortality with Emphysematous pyelonephritis vary from 7 to 75% [6,7] . It needs special attention because of its life-threatening potential.

Though it has generally been regarded as a rare infection, with the more extensive use of ultrasonography and computed tomography in the evaluation of patients with features of sepsis or complicated urinary tract infection (UTI), more cases of Emphysematous pyelonephritis (EPN) are being recognized. Huang et al believed that EPN is not rare and should be considered an important clinical entity [8].

Earlier days, emphysematous pyelonephritis was managed by nephrectomy or open surgical drainage and appropriate antibiotics[6]. Fluoroscopic guided percutaneous drainage for the treatment of emphysematous pyelonephritis was first described by Hudson et al [9]. With the advent of image guided drainage procedures and endoscopic drainage procedures - PCD, endoscopic stenting and medical therapy consisting of intravenous antibiotics and glycemic control measures are often applied. This is a disease that most commonly affects diabetics- a systemic disease with proven hazardous effect over the other uninvolved kidney in the long run. Moreover emphysematous pyelonephritis can be a bilateral problem in 10%[10] and can affect solitary kidneys. Renal conservation becomes more preferable in more instances.

# AIMS AND OBJECTIVES

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1. To study the possibility of renal salvagibility by minimally invasive approaches in emphysematous pyelonephritis.

2. To analyse the prognostic factors that favours renal salvagibility in emphysematous pyelonephritis

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

## Definition:

Emphysematous pyelonephritis is defined as a severe acute bacterial infection of the kidney characterized by the presence of gas within the renal parenchyma, collecting system or perinephric tissue [11].

Some investigators referred the term emphysematous pyelonephritis is renal parenchymal infection, whereas most prefer to include both conditions of renal parenchyma and perinephric tissues under the same designation[6]. The definition that is accepted is the possible manifestations of gas-forming acute renal infections [8].

## Sites:

Emphysematous pyelonephritis develops in the following sites of urinary tracts:

1. Emphysematous nephritis - involvement of renal parenchyma
2. Emphysematous pyelitis – gas in calyx and pelvis
3. Emphysematous ureteritis – collection of gas in ureter
4. Emphysematous cystitis – gas in urinary bladder [12].

## Etiology:

Emphysematous pyelonephritis exclusively occurs in diabetic patients, in patients not with DM it is due to obstruction of the reno ureteral sysyem [8,11,13,12].

As stated by Gillies and Flocks in 1941, three factors that are essential for gas formation are:

1. Urinary tract obstruction
2. Uncontrolled diabetes mellitus [11]
3. Gas producing organisms [6]
4. Defective immune system [6,14].

**Causative organisms:**

EPN is caused by an organisms that are normal habitants of urinary and gastrointestinal tracts. Based on the study by Michaeli et al [6], Escherichia coli was the most common organism (71%). In 19% of the cases >1 organism was present. *Aerobacter aerogenes* and *Proteus mirabilis* were isolated in some patients. Anaerobic bacteria were grown rarely [6].

According to Huang et al, pathogens was identified in 98% of cases. *E.coli* was the commonest organism isolated (69%), *K.pneumoniae* was the second (31%). 6% had *E.coli* infection along with *Streptococcus spp.* or *Proteus spp*. Anaerobic organisms were not obtained [8].Thus the most common organism grown is *E.coli* followed by *Klebsiella*. *Proteus, Pseudomonas,*

*Aerobacter aerogenes*, *Streptococcus* and rarely anaerobes, *Candida albicans* and *Cryptococcus* may be grown[15].

**Role of diabetes mellitus:**

Diabetic patients are immune compromised hosts and they have susceptibility for bacterial infection [14]. EPN most commonly occurs in diabetics. Diabetes mellitus was present in 87% of the emphysematous patients according to Michaeli et al. High tissue glucose levels acts as source for the organisms to produce carbon dioxide and hydrogen via the fermentation of sugar[6]. EPN is found exclusively in uncontrolled diabetics, still reported in non diabetics and diabetics with excellent diabetes control. In the non diabetic patients, disease is less extensive and is almost always associated with obstruction of reno ureteral unit [6].

**The role of obstruction**

Obstruction was present only in 40% of patients of EPN, Michaeli et al refutes the notion gas formation needs obstruction. Mostly, bilateral EPN and EPN in solitary kidneys had urinary tract obstruction. EPN nearly always is associated with ureteral obstruction in non-diabetics[6]. In Huang et al's study, 22% of diabetics and all the non diabetics (2 patients) had associated urinary tract obstruction. Left kidney than the right one more frequently is affected by urinary tract obstruction (64% vs 36%)[8].

**Pathophysiology**

It is said that hyperglycemia is source for the organisms to produce CO2 and hydrogen via the fermentation of sugar [6].

Two important features found commonly in EPN are necrotizing infection and compromised vascular supply shown by presence if thrombus in intra renal vessels and renal infarctions. Theory of Schainuck and associates supports these findings[16,14] emphasizing the importance of impaired tissue and vascular response. Obstruction and diabetic glomerulopathy are local factors and systemic factors such as increased risk of infectious complications associated with diabetes mellitus are all said to be responsible for tissue and vascular damage. Host response that predisposes to tissue damage is the impaired immune response, not the hyperglycemia.. The impaired host response theory is probable explanation for the presence of EPN in patients without diabetes and also in those without evidence of infection. In patients with diabetes mellitus and EPN both mechanisms (sugar fermentation and defective host response) coexists and explain the origin of profuse gas production[6].

**Cause of Gas Formation**

Metabolic energy is required in a constant manner by the growing organism. Fermentation proceeds via the glycolytic (Embden-Meyerhof) pathway, in which two molecules of adenosine triphosphate (ATP) are produced. Pyruvate is generated during the process of conversion of NAD+ to

NADH as end product. Apart from this several other pathways have been evolved in the microorganism for the reoxidation of NADH by pyruvate or its derivative such as lactic fermentation (*streptococcus*, *lactobacillus*), fermentation of alcohol The substance that is produced by *Enterobacteriaceae spp.* in mixed acid fermentation is stable in non acidic pH. However the fermentation reactions lead to pH 6 or below due to accumulation of acids. Gas forming microorganism like *E.coli,* will form an enzyme formic hydrogenylase. Formic acid is converted into carbon dioxide and hydrogen by Formic hydrogenylase. The result of mixed acid fermentation is production of hydrogen. None of the other 5 pathways listed above would give rise to hydrogen gas as the end product. Gas composition of a gas bubble tends to equilibrate naturally with the surrounding tissue, hence it is reasonable that the gas will contain reasonable amounts of nitrogen, as well as oxygen, carbon dioxide and hydrogen. Trace amounts of ammonia and methane might arise from the fermentation of mixed amino acids that were produced by the degradation of the necrotic tissue[14].

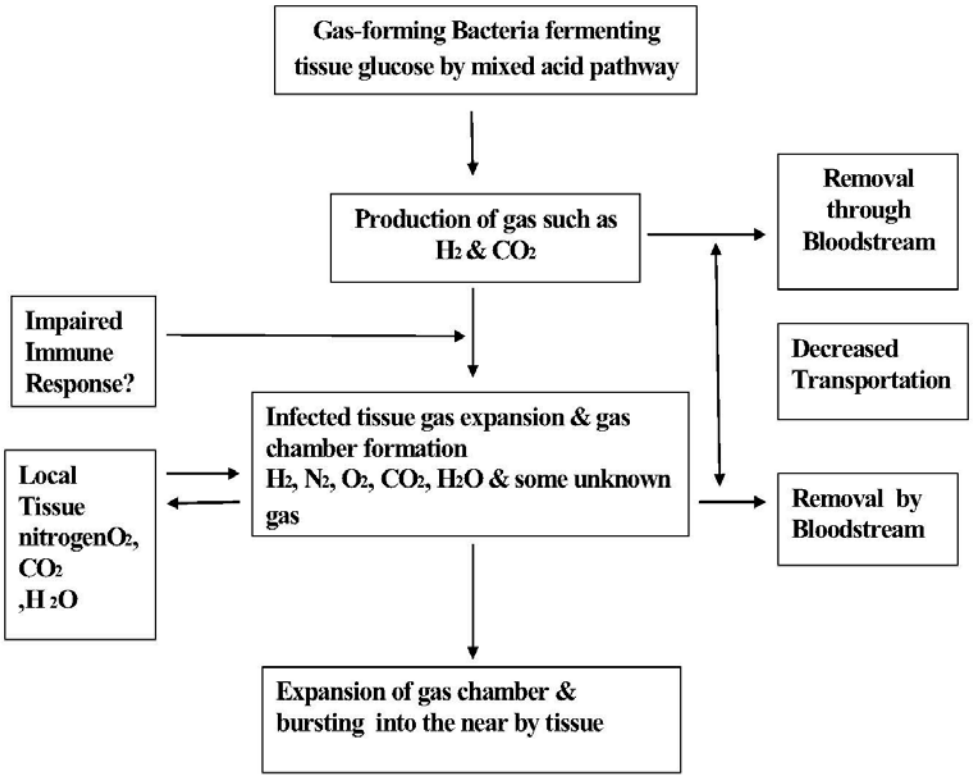
The mechanism of gas formation is proposed to be due to various causes like increased gas formation, delay in clearance of gas by vascular compromise, gas chamber formation, and the expansion or collapse of the gas chamber.

Factors that may be involved in the pathogenesis of EPN are four in number namely gas forming bacteria, high tissue glucose levels, impaired tissue

perfusion, and a defective immune response. Hyperglycemia is the cause in diabetes mellitus[14,17,18]. The unrelieved obstruction in case of urinary tract because of back pressure and impaired circulation, results in decreased transportation of gas and subsequent creation of a gas chamber(ie, EPN)[8].

Local tissue damage would markedly interferes with clearance of end products away from the lesion and accumulation of gas occurs. In the non-diabetic also, glucose may serve as the substrate for gas formation. In normal non-diabetics, around 20 mg% of glucose may be present and up to 60 mg% with acute or chronic renal disease. It is said subclinical glucosuria in renal infection may some times be enough to generate sufficient amounts of gas [12 to 36 cc of gas from 100 mg glucose at S.T.P] [2].

**Fig 1 : Pathogenesis behind emphysematous urinary tract infection[14]**



## PATHOLOGY

1. Features of inflammation of pelvis and parenchyma,
2. Abscesses in cortical region of kidney
3. papillary necrosis.
4. changes due to decreased tissue circulation – infarction, thrombosis of vessels, arteriosclerosis and glomerulosclerosis.
5. Infiltration of cells associated with inflammation, focal necrosis and abscess formation.
6. Kimmelstiel-Wilson nodules, hyalinized arteriosclerosis and glomerulosclerosis – which are features of diabetic nephropathy.

The inflammatory findings are limited to the kidney in class 2 EPN, in extensive involvement extension to the perinephric areas in seen.

## CLASSIFICATION

**Huang et al Classification**.

**Class 1** – Gas in the collecting system only (Emphysematous pyelitis)

**Class 2** – Gas in the renal parenchyma without extension into the extrarenal space

**Class 3A**- Extension of gas or abscess to the perinephric space

**Class 3B**- Extension of gas or abscess to the pararenal space

**Class 4** – Bilateral EPN/Solitary kidney with EPN [8]

**Wan et al**

**Type I**

Renal necrosis with either the presence of a streaky/mottled gas pattern demonstrated on radiograph or total absence of fluid content on CT or CT with lung window display.

**Type II**

Characterized either by the presence of gas in the collecting system or the presence of renal/perirenal fluid in association with a bubbly/loculated gas pattern.

Type I emphysematous pyelonephritis is associated with more extensive parenchymal necrosis and a more fulminating clinical course than type II [7]

**Mitra et al also divided** renal emphysema into two distinct entities and proposed to have important prognostic and therapeutic implications.

1. **Emphysematous pyelitis** : A milder form is characterised by gas in the pelvicalyceal system. It is associated with obstructive uropathy[6,19].
2. **Emphysematous pyelonephritis : Gas in this type extends in addition to** renal pelvis, calyx, parenchyma, tissues around kidney and retroperitoneum [15.]

**Michaeli et al classification**

**Stage I** - Gas within the renal parenchyma or in the perinephric tissues.

**Stage II** - Presence of gas in the kidney and its surrounding tissues.

**Stage III** - Extension of gas through fascia of kidney or Involvement of bilateral EPN.

**CLINICAL PRESENTATION**

EPN presents commonly in adults [20].Juvenile diabetics is not a risk factor. Men are less affected than women [21]. The patient presents with features of pyelonephritis that fails to resolve during the first 3 days of treatment. Chronic infection preceding an acute attack is also seen in some cases. Almost all patients present with any two of the classic triad of fever, vomiting and flank pain and pyuria.[16]. May also show symptoms non-specific, such as pain in the abdomen, nausea, vomiting and altered sensorium. It accompanies shock, lumbar tenderness, dysuria, crackling feel in the flanks, and pneumaturia.[2],[3],[4],[6],[9] When infection involves the collecting system pneumaturia is present. Culture test of urine are invariably positive.

Most often organism identified is E.coli; Klebsiella and Proteus are less common. Huang et al founded fever in 79%, nausea/vomiting in 17%, shock in 29%, alteration in conscious level in 19% and acute renal functional impairment in 35% of cases [8].

Michaeli, in his study of 55 patients found chills, fever(57%) , flank pain(46%) , Lethargy and confusion(22%) , Nausea preceding vomiting (14%), shock and coma(14%) . Pyrexia of unknown origin was the presenting feature in some 18%. Pneumaturia is uncommon. The average duration of all these symptoms was 21 days- the range being 0.5 to 240 days before diagnosis [6].

Diabetes has higher incidence in EPN - 80% (Shokeir et al [12]), 96% (Huang et al [8]), and 87% (Michaeli et al [6]).[25] The preferable side of involvement was predominantly left (60% in Shokeir et al's study[12] and 47% (Bum Soo Park et al)[5]. Affection of both kidneys also ranged from 5% [12] to 20%[22]. The clinical sign is costovertebral angle tenderness[5]. Leukocytosis present in about 67% and thrombocytopenia is seen in 46%[8]. Patients may present in medical emergency casuality viz., diabetic ketoacidosis.

**MICROBIOLOGY**

The commonest reported organism is E. Coli (69% to 73%) followed by Klebsiella (27%)[8,6]. Blood stream involvement by bacteria is found in almost 50% of all the cases and usually the organisms that are grown in urine , blood and tissue cultures matches it [8,12]. 19% of cases show more than one organism[6].

**RADIOLOGY**

The definitive mode of diagnosis of EPN is by radiology. Radiology helps to confirm the presence of EPN and classify EPN hence guiding the

treatment and assessment of prognosis of the disease. It is investigated by X-ray, USG and CT scan. Best early pick up rate is by CT. Extent of gas dissemination is defined with great accuracy. It stages the gas distribution and facilitates description of the gas in the renal system as streaky, mottled, bubbly, rim like, crescent shaped, locular, etc. CT helps in diagnosis and follow up. It picks up the development of new lesions as well, resolution of the gas and abscesses.

The USG, though not sensitive in identifying renal gas helps in diagnosis obstruction of urinary tract. Intraparenchymal gas is shown as strong focal echoes[23,24. 8] The "dirty" white shadows is diagnostic whereas "clean" shadowing is due to calculus. USG is cheap, readily available and non invasive.

Plain X-ray of the KUB region shows gas distribution in the renal and perirenal areas in 33% of cases. One may differentiate renal gas from overlying intestinal gas in equivocal cases by Infusion nephrotomography [6]. Differential diagnosis of gas in EPN is traumatic renal infarction

IVU [Intravenous urogram] demonstrates absence of excretion of kidney in 27 around 45%. Even those who showed excretion, had poor delineation. Due to the toxic side effects of contrast in IVU on the kidneys in diabetics and as not much information is provided by it because of non functioning or poorly functioning system on affected side, its use should be weighed judiciously

compared to plain CT. IVU shows gas and other findings such as areas of renal inflammation like indistinct margins and mass effect[12].

Obstruction is demonstrated in around 25% of cases [21] and is better demonstrated by USG or retrograde pyelogram. 3 main patterns of findings on X-rays described by Langston and Pfister had an apparent similarity with the stage of the disease. Earliest sign is diffuse mottled appearance of the renal parenchyma, with radial distribution of the gas bubbles either along the pyramids or within the tubules. Bubbly parenchyma with crescent of gas denotes further clinical deterioration. With extension through the Gerota's fascia, gas can be seen in the retroperitoneum and may even extend upto the posterior thoracic wall[25].

Bum Soo Park et al found plain Xray KUB as reliable modality of screening investigation (picked up 50% of cases) and CT as the most reliable modality for diagnosis confirmation (Diagnostic rate 100%) for planning treatment. According to him USG unhelpful to locate renal gas[5].

**MANAGEMENT**

All patients invariably needs supportive resuscitative measures as patient is acutely ill. Intensive measures aimed at glycaemic control by insulin , maintenance of fluid balance by serial electrolyte level monitoring and treatment of shock as quickly as possible. Antibiotics can be changed later according to sensitivity once culture results are available but beforehand

empirical antibiotic is started immediately. Obstruction, if present, should be relieved urgently.

Joseph.B.Stokes JR[26], Dunn and Dewolf et al, in their study of 3 cases treated by nephrectomy[27] favoured an initial trial of conservative management with antibiotics. According to Schultz and Klorfein, the disease is best managed by intensive medical methods of treatment and is not an indication of emergency surgery. They found that involvement of contralateral kidney was often present and really nephrectomy is unwarranted[2]. Their main concern was the probability of recurrent disease in the contralateral kidney.

Nephrectomy could be considered if the renal and perirenal gas or the toxic symptoms persists inspite of conservative management. Such patients may even be started on lifelong suppressive antibiotics and be followed up strictly instead of nephrectomy. Medical management of EPN is preferable because of high chances of involvement of opposite kidney due to disease recurrence as well as diabetic disease. Avoidance of surgery, vigorous blood sugar control, appropriate antibiotics and relief of obstruction was rational.

Previous days treatment ideas were that mere medical treatment does not decrease mortality or morbidity hence supplemented by prompt surgical drainage and nephrectomy. Renal conservation has come into vogue for reasons already mentioned. The need to save the kidney in the setting of a high probability of the disease occuring in the opposite side later as well as the long

term effects of diabetes on the opposite kidney. In conditions like synchronous bilateral disease as well as EPN affecting a solitary kidney this modality of minimal invasive procedures with renal conservation is highly desirable.

The importance of perinephric extension of gas was insisted by Huang et al[8]. Even though the differences in clinical presentation is not that much different between classes, mortality and failure of PCD tends to increase from class 1 to 4. The best prognosis was enjoyed by class 1 patients. PCD and antibiotics with relief of obstruction keeps these patients recovering.

In class 2 also, all patients treated so, were shown recovery. In class 3 and 4 morbidity is based on the presence of risk factors. Presence of less than two risk factors response increased when subjected to PCD and antibiotic. 85% of patients with <2 risk factors (reduced platelet count, decreased renal function, alteration in consciousness and low blood pressure) show renal conservation. The failure rate of conservative treatment (i.e., combined medical and minimally invasive treatment) was less for those with no or single factor example 15% for those with no or a single risk factor and 92% for those with 2 or more risk factors. In such cases, nephrectomy is expected to give the best management outcome. The usefulness of PCD is it drains the pus, releases the gas and thereby the pressure to local circulation is relieved and improves circulation, provides pus that can be cultured and can help in further management and can promote increased rates of success in extensive EPN.

They suggest PCD and antibiotics less extensive disease (class 1 & 2) and also for extensive EPN with < 2 risk factors. This leads to a renal conservation in most of the cases.

Nephrectomy provided the best treatment outcome for extensive EPN with fulminant course (2 or more risk factors). Even in class IV drainage of gas is attempted first. Emergency nephrectomy in such individuals carries poor prognosis and increased mortality. Nephrectomy should be done if PCD fails.

Prognostic factor do not include poor glycemic control. Multiple organ dysfunction in EPN affects outcome of treatment as disease runs a rapid progression with poor outcome. Severe proteinuria also carried poor outcome and seemed to be involved in extensive disease. The causes of severe proteinuria may be multifactorial with fever underlying glomerulonephritis, and diabetic nephropathy may contribute.

Michaeli et a[16], in their review, is of the opinion that renal conservation were often not successful whereas bilateral surgery was successful at times. Thus most important factor associated with survival was an approach combining medical and surgical treatment. The observation is the most favourable outlook of minimally invasive surgery was for individuals with non obstructive unilateral disease and short interval of symptoms of EPN.

Wan et al described two classes of EPN[7]. The dry type (type 1) which is associated with parenchyma destruction, absence of fluid collection and streaky

or mottled gas presented a fulminant course with a mortality rate of 66%. Type 2 is associated with a mortality rate of 18%. This difference in observation is due to compromise in immunity and vascular insufficiency in the kidneys and immunocompromise in the diabetics. They described serum creatinine > 1.4mg% was associated with a poor outcome.

In their study of 20 cases, Shokeir et al[12] is in for nephrectomy which is scheduled immediately follow aggressive resuscitation and diabetes control. Stein et al, in their case report and review of literature[22], insisted on treatment of bilateral disease in multiple ways and combination. Endoscopic stenting, PCD bilaterally / unilaterally which includes PCN both sides or single side depending on extensive involvement and stenting. Nephrectomy ipsilateal and open drainage decided according to progress.

Few others also considered nephrectomy to be the most effective modality of treating EPN as evidenced by Bum Soo Park et al[5] . They were for concomitant immediate nephrectomy and all supportive and resuscitative measures. Renal conservation (with PCD and antibiotics) were aimed in solitary kidney, poor general/medical condition rendering the patient unfit for surgery, inadequate function of other kidney and disease of bilateral kidney. Nephrectomy is done through 11th rib bed approach through the loin.

Hung et al [29] have noted anaerobic bacteria, B. fragilis as the causative organism in a case of EPN. According to him anaerobes are never a causative

pathogen rarely one case has been reported with clostridium[30]. Anaerobes also cause hence anaerobic treatment is needed.

# MATERIALS AND METHODS

# MATERIALS AND METHODS

**Study group:** Patient who were admitted in Kilpauk Medical College and Government Royapettah Hospital with Emphysematous Pyelonephritis were included in the study.

**Study design:** Prospective clinical study

**Materials:** Patients who had features of emphysematous pyelonephritis were admitted, investigated and descriptive study made with relation to age, sex, diabetic status, level of consciousness, shock, biochemical parameters, imaging studies and subjected for conservative and minimally invasive modalities of treatment. Results were analysed risk factors which led to invasive treatment were also analysed

**Study period:** 2 years from December 2012 to November 2014

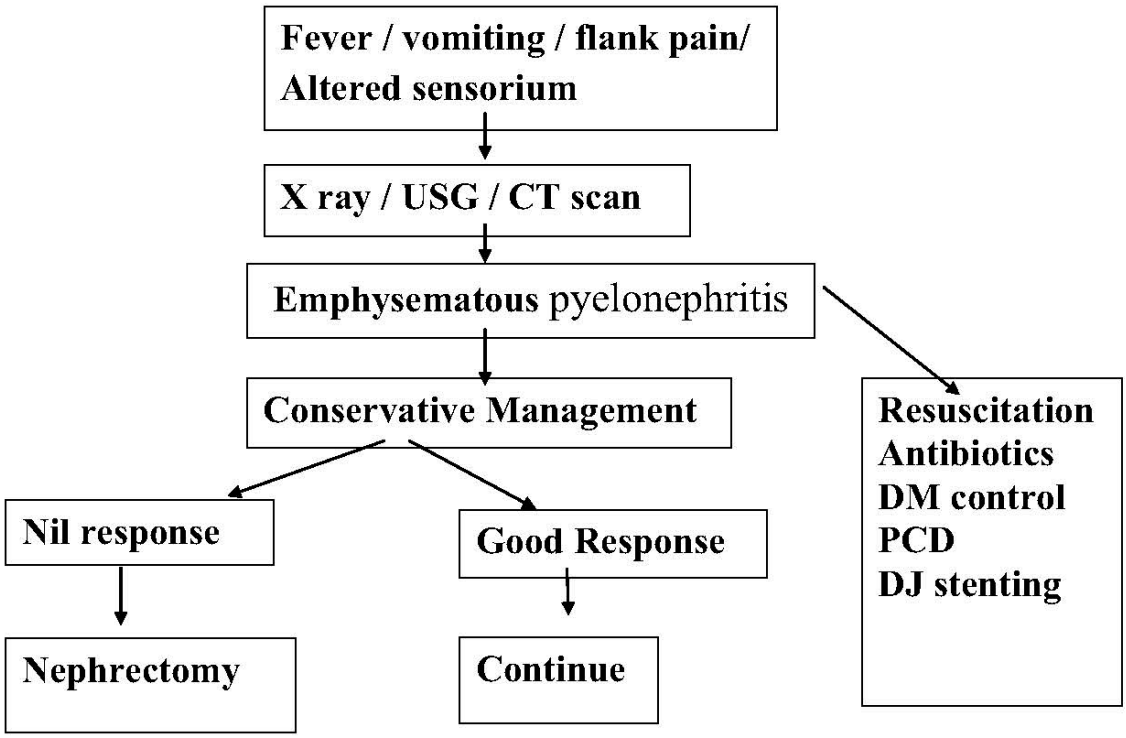
**Inclusion criteria**

1. Patients with features of acute pyelonephritis with gas in the renal parenchyma and perinephric and paranephric tissues. The symptoms were Fever, chills, loin pain, altered sensorium and vomiting.
2. Patients admitted in the emergency, but subsequently evaluated and found to have gas in the renal parenchyma and beyond it with features of acute pyelonephritis

**Exclusion criteria**

1. Patients with clinical features suggestive of acute pyelonephritis without gas in the renal parenchyma.
2. Presence of history of recent endoscopic or open interventions in the urinary tract.
3. Recent catheterization history.

**Fig 2: Protocols of Management**



All patients who had symptoms of fever, loin pain and vomiting, altered sensorium underwent preliminary Xray KUB and USG abdomen. If findings suspicious of gas were present, they are subjected for CT scan for confirmation and basic staging of class (with contrast enhancement if the renal parameters were not raised). Patients in whom gas could not be noticed in either of these investigation also underwent CT scan based on clinical examination and suspicion due to clinical features of acute toxic illness. The patients were stratified based on Huang et al's CT classification[8].

On admission, baseline history recorded which included age, sex, history, duration of symptoms and diabetic mellitus status, its duration, modality and regularity of treatment. The clinical features recorded to know whether symptoms and signs have got significance in assessing outcomes of minimally invasive interventions in management of EPN. Signs included hemodynamic status, presence of shock if any, the degree of consciousness, hydration status abdominal distension and mass if any. Basic Investigations like urine culture, blood culture, blood glucose level, serum creatinine, blood urea, total and differential WBC counts, blood haemoglobin level, urine acetone and serum electrolytes when there is raise in renal parameters were recorded on admission. A blood platelet count was done.

Shock was defined as systolic BP <90 mm Hg. Raised renal parameter was defined as increased if serum creatinine > 1.5 mg% or blood urea >40mg%. Altered consciousness was defined as patient in confusion, delirium, stupor or coma.

All admitted patients were given resuscitative measures to correct hydration, urosepsis and relief of obstruction. Antibiotic chosen for all patients belong to 3rd generation cephalosporins (cefotaxime, ceftriaxone or cefoperazone), piperacillin tazobactum and metrogyl. Aminoglycosides may be added after looking into renal parameters if normal. Antibiotics were later changed if necessary, based on culture and sensitivity. Intensive resuscitation was carried out with hydration, correction of electrolyte imbalance if any and measures to treat diabetes was initiated with insulin.

Initially patients are subjected to undergo conservative management with only antibiotics, antibiotics with PCD &/or DJ stenting. PCD was defined as percutaneous aspiration/ drainage of pus and gas with/without percutaneous nephrostomy. PCN/ PCD was done usually under USG guidance using a 8.5Fr single puncture PCN catheter in prone, prone oblique or lateral positions via the flank taking strict aseptic care to avoid contamination of the peritoneum.

Failure of minimal invasive treatment is continuing symptoms and signs with persistence of gas in radiological images. Duration of time to recovery from acute illness recorded for all patients. Assessment of response done by CT, USG

and serial measurement of platelet count, total count, blood urea, serum creatinine and if on PCN, PCN fluid analysis. Our patients, were stratified depending on outcome, as “good” and “poor” outcome . The “good” outcome is those treated with antibiotics only or PCD +/_ DJ stenting or DJ stenting only with antibiotics. The “poor” outcome is who failed to respond to minimal invasive treatment and needs invasive open drainage/nephrectomy to prevent mortality.

**Statistical analysis:**

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with the Unpaired t test and categorical variables were analysed with Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using EpiInfo software (7.1.0.6 version; Center for disease control, USA) and Microsoft Excel 2010.

**Sample size calculation**

Sample size was determined based on

**Study**

Emphysematous pyelonephritis: Our experience with conservative management in 14 cases

**Authored by**

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**Published in**

Urol Ann. 2013 Jul-Sep; 5(3): 157–162.

In this study two (14%) had thrombocytopenia while seven had deranged renal parameters at the time of admission

**Description:**

• The confidence level is estimated at 95%

• with a z value of 1.96

• the confidence interval or margin of error is estimated at +/-12

• Assuming that the sample will have the specified attribute p% =14 and q%=86

$n = p\% \times q\% \times [z/e\%]^2$

$n= 14 \times 86 \times [1.96/12]^2$

n= 32.12

Therefore 32 is the minimum sample size required for the study

In our study we have taken 40 as the sample size

(n=636 in good outcome Group and n=4 in bad outcome Group)

# RESULTS

# RESULTS

**Sample size:**

Total number of patients included in this study was 40.

**Age:**

The mean age was 55.02 yrs with a standard deviation of 7.76. Age was not significantly related to the outcomes in our study (p=0.48474) since p>0.05

Fig :1

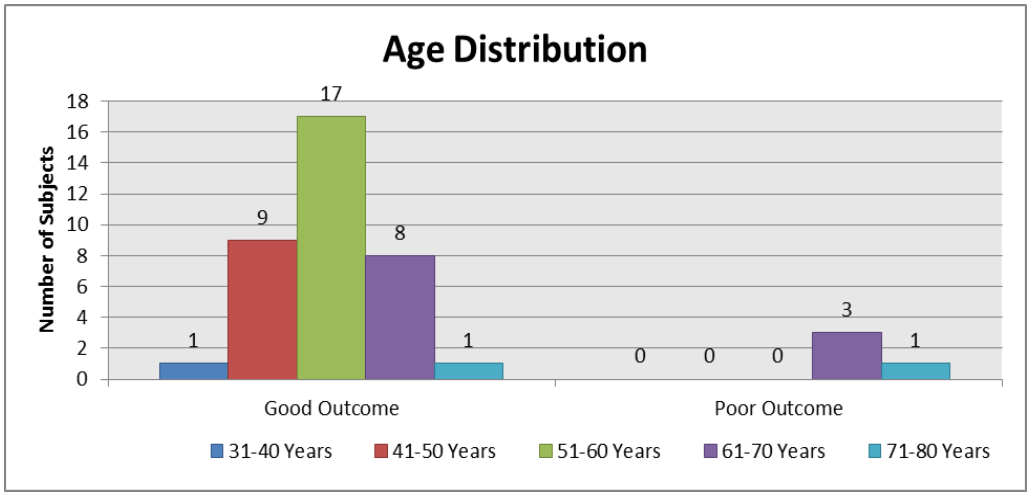



**Figure 1: Age distribution**

| Age | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **31-40 Years** | 1 | 2.78 | 0 | 0.00 |
| **41-50 Years** | 9 | 25.00 | 0 | 0.00 |
| **51-60 Years** | 17 | 47.22 | 0 | 0.00 |
| **61-70 Years** | 8 | 22.22 | 3 | 75.00 |
| **71-80 Years** | 1 | 2.78 | 1 | 25.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Age | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 55.02778 | 66.75 |
| **SD** | 7.766176 | 7.675719 |
| | **P value Unpaired t test** | 0.48474 |

**Sex:**

25% of the total cases were males and 75% were females. Age and Sex distribution are not statistically significant, it means that there is no difference between the Outcome groups.

In other words the groups contain subjects with the same basic demographic characteristics. (p=0.5558) Fig: 2

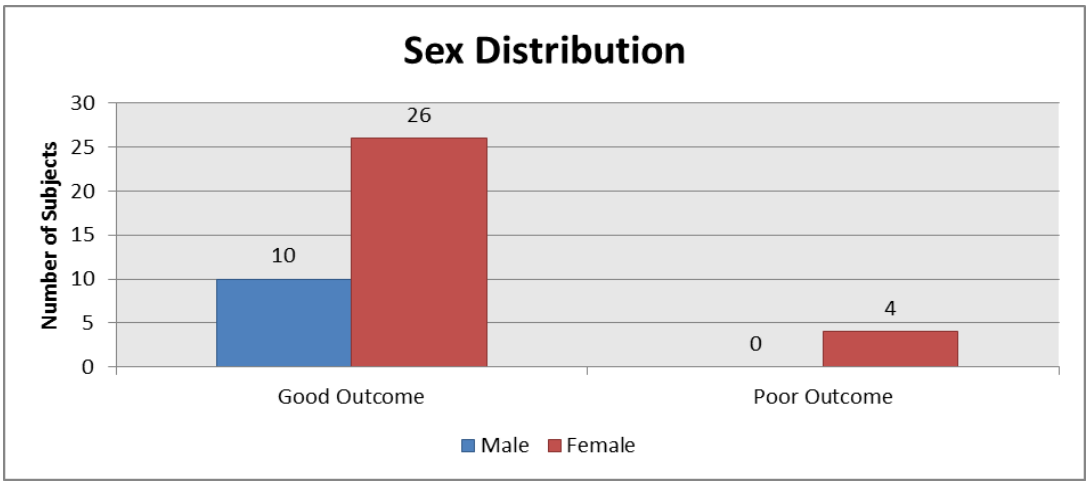



**Figure 2: Sex distribution**

| Sex | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Male** | 10 | 27.78 | 0 | 0.00 |
| **Female** | 26 | 72.22 | 4 | 100.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.5558 | |

**Frequency of side affected**:

The left kidney was affected in 52.50% of the cases, the right kidney in 40% of the cases and both kidneys in 7.50% cases.

By conventional criteria the association between Sides affected and Outcome groups not statistically significant since p > 0.05 (p=0.2057) (Fig 3).

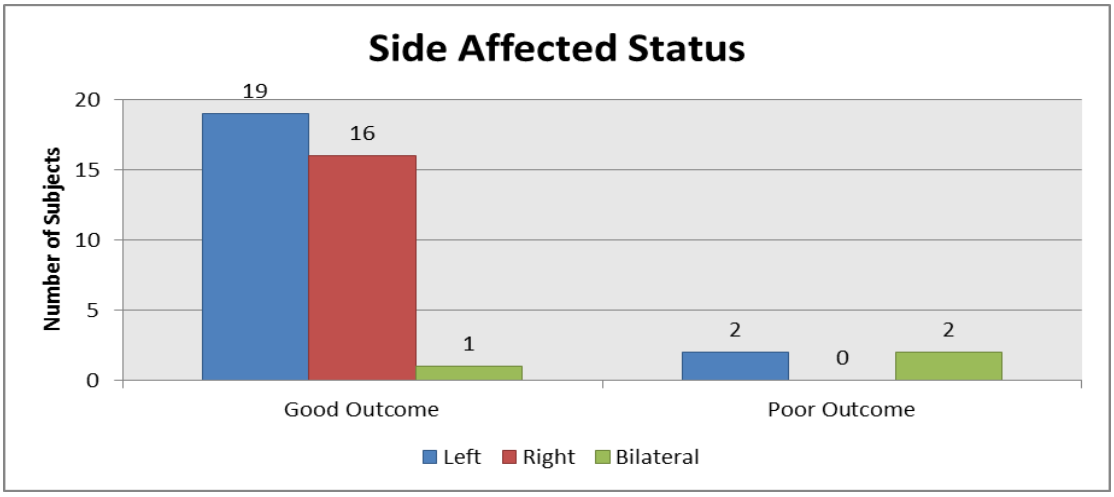



**Figure 3: Frequency of side affected**

| Side Affected | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Left** | 19 | 52.78 | 2 | 50.00 |
| **Right** | 16 | 44.44 | 1 | 25.00 |
| **Bilateral** | 1 | 2.78 | 1 | 25.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.2057 | |

**Associated Diabetes mellitus:**

87.5% of the patients were diabetic of which 8.5% were newly detected.12.5% of the patients were non diabetic.

By conventional criteria the association between Diabetic status and Outcome is not statistically significant since $p > 0.05$ ($p=0.9999$) (Fig 4).

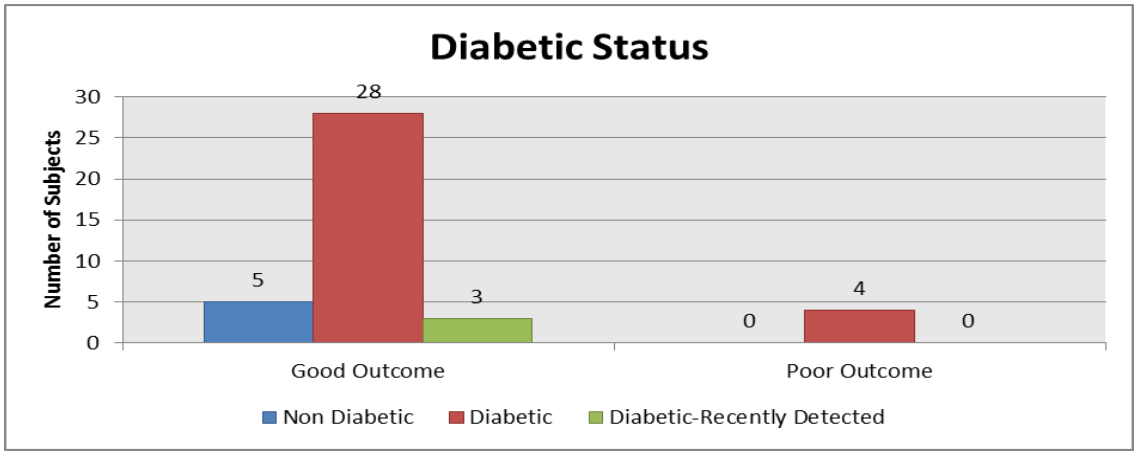



**Figure 4: Diabetic status**

| Diabetic Status | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Non Diabetic** | 5 | 13.89 | 0 | 0.00 |
| **Diabetic** | 28 | 77.78 | 4 | 100.00 |
| **Diabetic-Recently Detected** | 3 | 8.33 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.9999 | |

**Treatment of Diabetes mellitus:**

Of the diabetics, 65% were on regular treatment, 25% of whom no treatment status available and 5% of patients were on irregular treatment.

By conventional criteria the association between Diabetes treatment and Outcome is not statistically significant (p=0.6446) (Fig5).

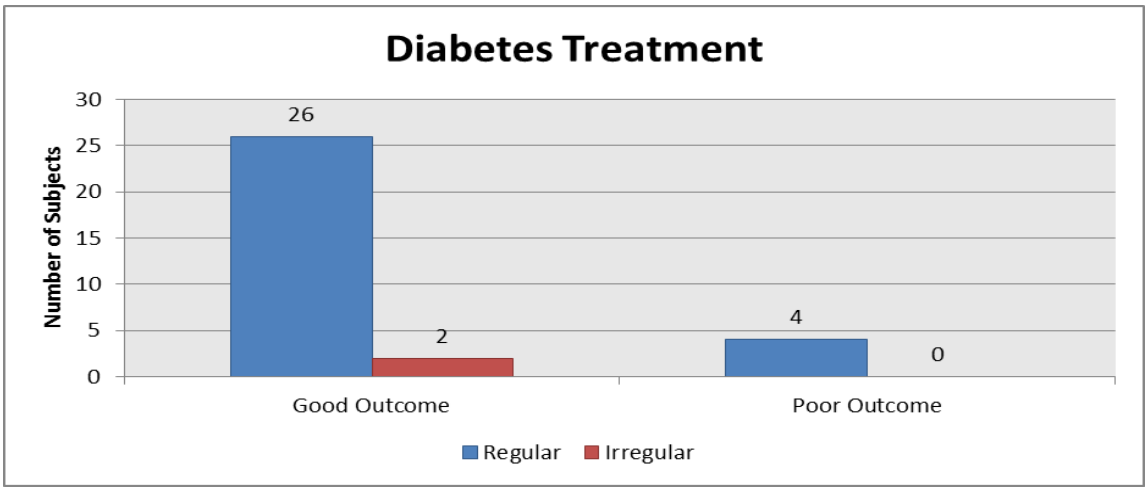



**Figure 5: Diabetes treatment**

| Diabetes Treatment | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Regular** | 26 | 72.22 | 4 | 100.00 |
| **Irregular** | 2 | 5.56 | 0 | 0.00 |
| **Unknown** | 8 | 22.22 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.6446 | |

**Mode of treatment of Diabetes Mellitus:**

65% were on regular treatment, of which 65% were on OHAs and 5% were on insulin.

By conventional criteria the association between Diabetic mode of treatment and outcome is considered statistically significant as p > 0.05 (p=0.6415) Fig :6

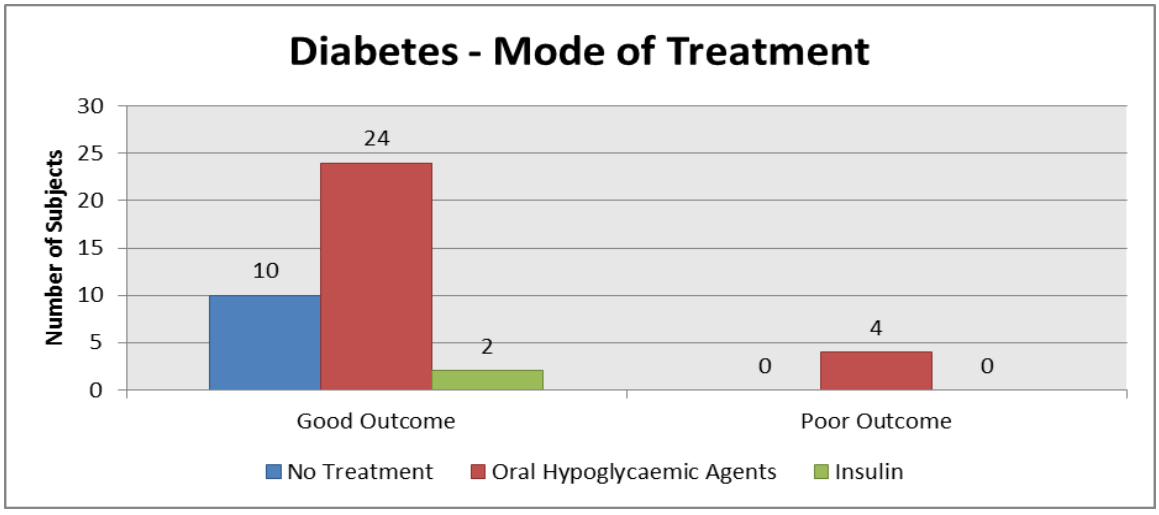



**Figure 6: Mode of Treatment of DM**

| Diabetes - Mode of Treatment | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **No Treatment** | 10 | 27.78 | 0 | 0.00 |
| **Oral Hypoglycaemic Agents** | 24 | 66.67 | 4 | 100.00 |
| **Insulin** | 2 | 5.56 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.6415 | |

**Duration of treatment of diabetes:**

Duration of diabetic treatment preceding occurrence of Emphysematous Pyelonephritis does not show significance with outcome of the disease.

By conventional criteria the association between Diabetes duration of treatment and Outcome groups is considered not significant (p=0.05836) Fig :7.

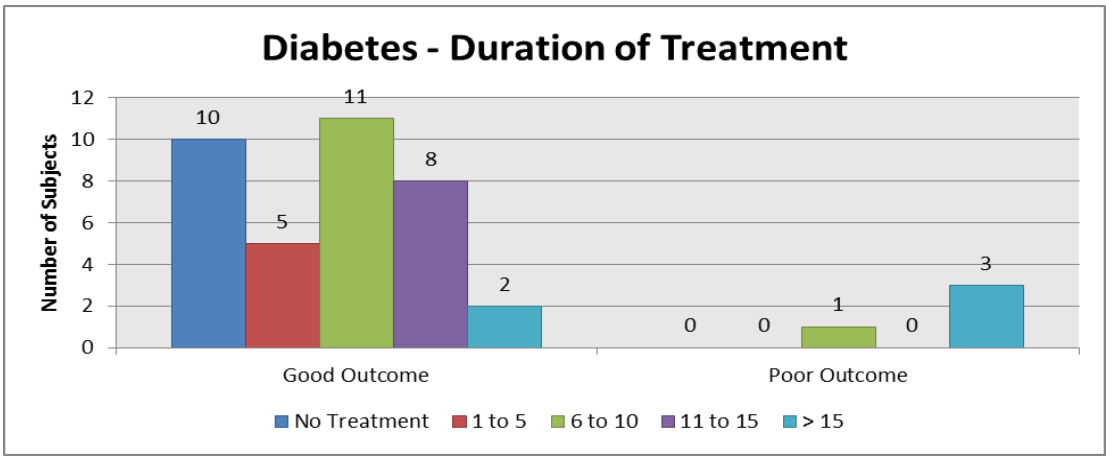



**Figure 7: Duration of Treatment of DM**

| Diabetes - Duration of Treatment(In Years) | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **No Treatment** | 10 | 27.78 | 0 | 0.00 |
| **1 to 5** | 5 | 13.89 | 0 | 0.00 |
| **6 to 10** | 11 | 30.56 | 2 | 50.00 |
| **11 to 15** | 8 | 22.22 | 0 | 0.00 |
| **> 15** | 2 | 5.56 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Diabetes - Duration of Treatment(In Years) | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 7.083333 | 14 |
| **SD** | 5.827889 | 4.898979 |
| **P value Unpaired t test** | | 0.05836 |

**Symptoms:**

The most common mode of presentation was fever & loin pain (26/40 ; 65%).Loin pain was the only presentation in 30% (12/40) Other modes of presentations like seizures, altered sensorium or vomiting constituted the rest (5%). Fig: 8

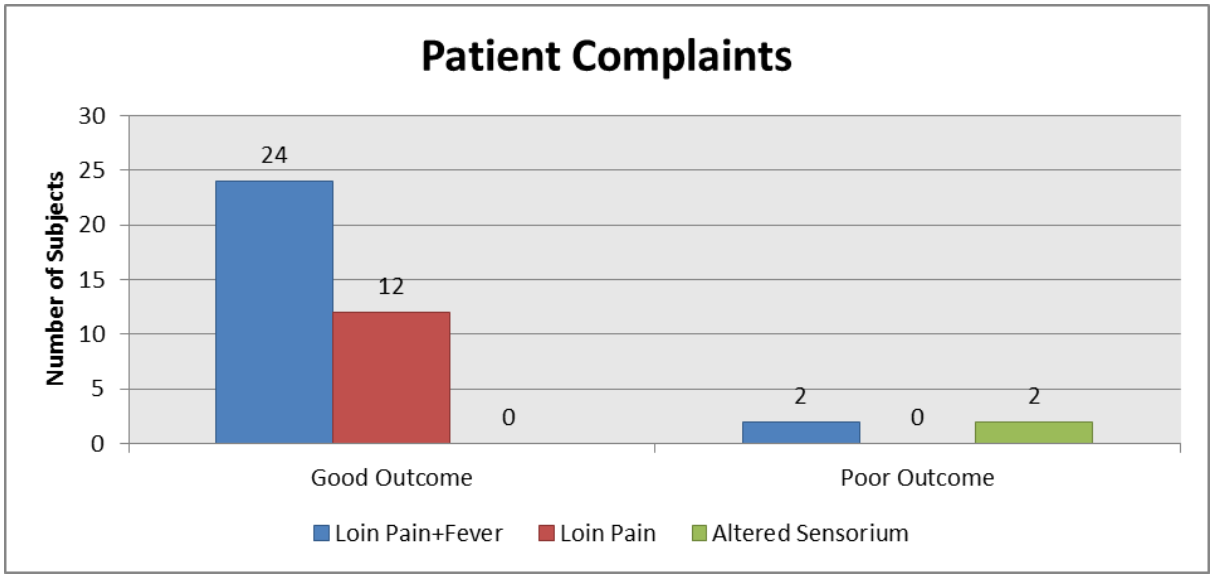
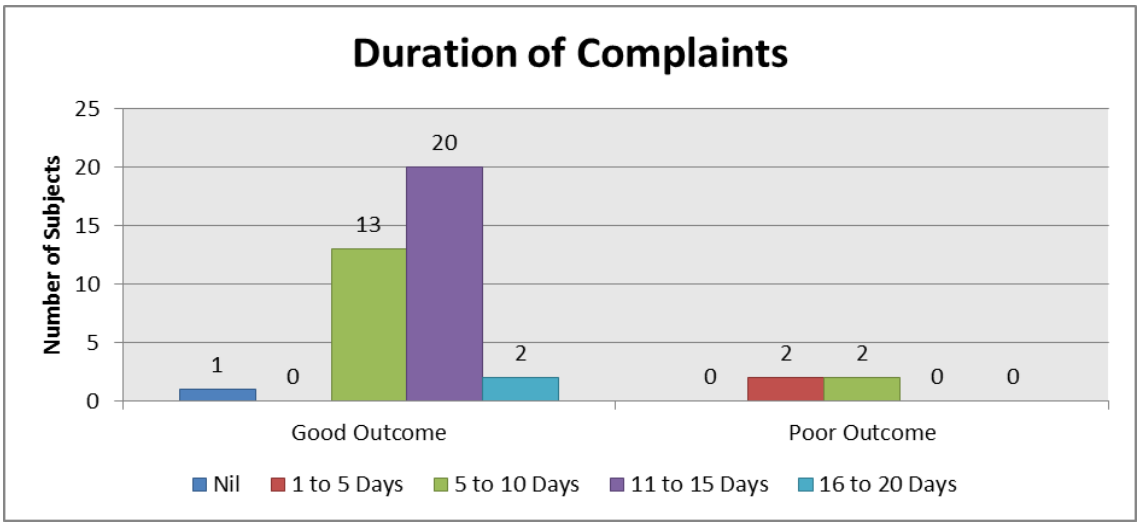



**Figure 8: Patient complaints**

| Patient Complaints | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Loin Pain+Fever** | 24 | 66.67 | 2 | 50.00 |
| **Loin Pain** | 12 | 33.33 | 0 | 0.00 |
| **Altered Sensorium** | 0 | 0.00 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

**Duration of complaints:**

Duration of complaints is short for poor outcome compared to good outcome. Mean duration is 11.52+/- 3.45 days for good outcome and mean duration for poor outcome is 6.25+/- 3.75




| Duration of Complaints | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Nil** | 1 | 2.78 | 0 | 0.00 |
| **1 to 5 Days** | 0 | 0.00 | 2 | 50.00 |
| **5 to 10 Days** | 13 | 36.11 | 2 | 50.00 |
| **11 to 15 Days** | 20 | 55.56 | 0 | 0.00 |
| **16 to 20 Days** | 2 | 5.56 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Duration of Complaints | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 11.52778 | 6.25 |
| **SD** | 3.451593 | 2.753785 |
| **P value Unpaired t test** | | 0.02286* |

**Findings on Clinical Examination:**

On clinical examination, the commonest finding was loin tenderness (75%), 20% presented with an abdominal mass and 5% with abdominal distension. Fig :9

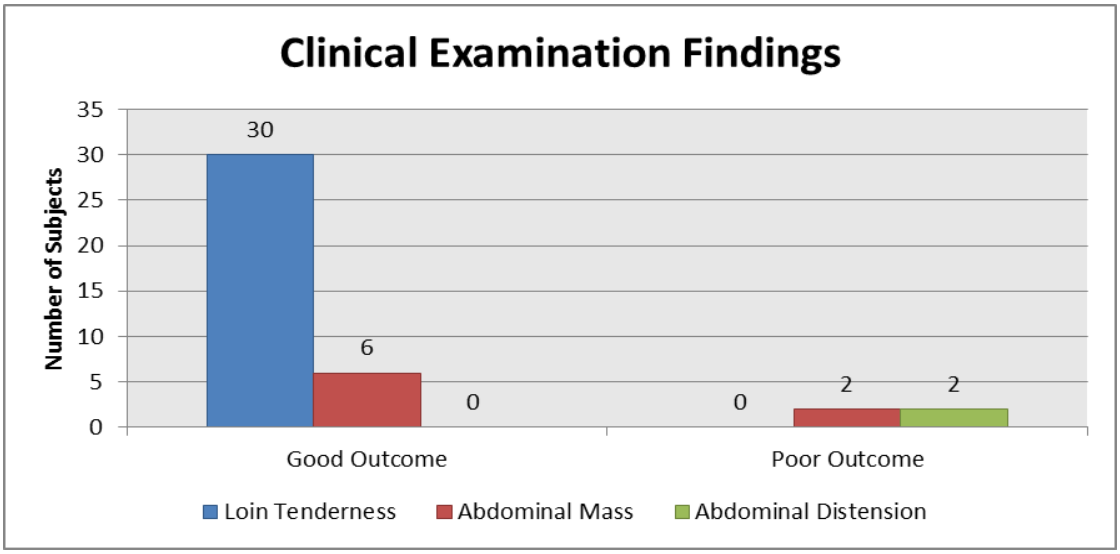



**Figure 9: Clinical Examination**

| Clinical Examination Findings | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Loin Tenderness** | 30 | 83.33 | 0 | 0.00 |
| **Abdominal Mass** | 6 | 16.67 | 2 | 50.00 |
| **Abdominal Distension** | 0 | 0.00 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

**Serum Creatinine:**

20 out of 40 patients (50%) had raised renal parameters. The rest (50%) had normal renal parameters. The relationship of serum creatinine value with the outcome reached statistical significance (p=0.001902 ) (Fig 10)

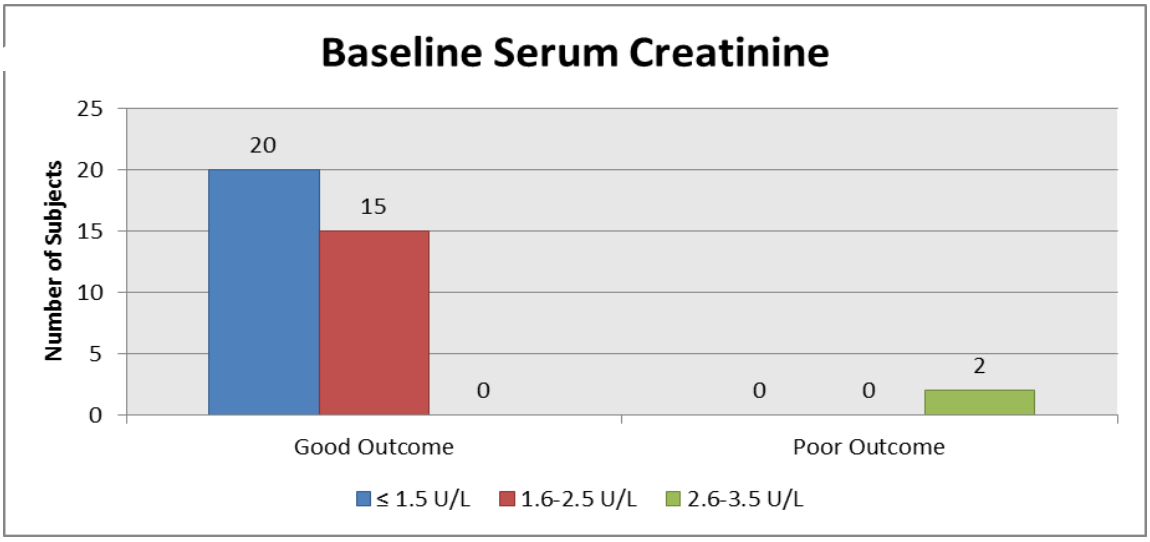



**Figure 10: Serum Creatinine**

| Baseline Serum Creatinine | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **≤ 1.5 U/L** | 20 | 55.56 | 0 | 0.00 |
| **1.6-2.5 U/L** | 15 | 41.67 | 0 | 0.00 |
| **2.6-3.5 U/L** | 0 | 0.00 | 2 | 50.00 |
| **3.6-4.5 U/L** | 1 | 2.78 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Baseline Serum Creatinine | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 1.447222 | 3.625 |
| **SD** | 0.585777 | 0.485627 |
| **P value Unpaired t test** | | 0.001092* |

By conventional criteria the association between Baseline Serum Creatinine levels and outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

This indicates that there is a true difference among groups and the difference is significant. In simple terms, when studying the Role of minimally invasive approaches for renal salvagibility in management of Emphysematous Pyelonephritis, the average serum creatinine levels in good outcome (1.48 U/L) is predominantly less when compared to bad outcome (3.63 U/L). It is statistically significant with a p-value of 0.001092 according to unpaired t test.

**Clinical Significance**

The average serum creatinine levels in good outcome group are meaningfully less than bad outcome group by 2.5 times with a mean difference of 2.18 U/L. Similarly the prevalence of normal serum creatinine levels are much more in good outcome group(55.56%) and prevalence of increased serum creatinine levels is much more in poor outcome group(100%)

This difference is true and significant and has not occurred by chance.

**Conclusion**

We conclude that there is meaningfully real increase in risk of developing poor outcomes in persons with increased serum creatinine levels among our study subjects. In short increased serum creatinine levels correlate with poor outcomes in our study subjects.

**Base line Blood Urea**

The blood urea values in the good outcome group was 41.416 +/- 8.6499 and in the poor outcome group was 99.5 +/- 9.036. The relationship of Blood Urea value with the outcome reached statistical significance (p=0.000435) (Fig11)

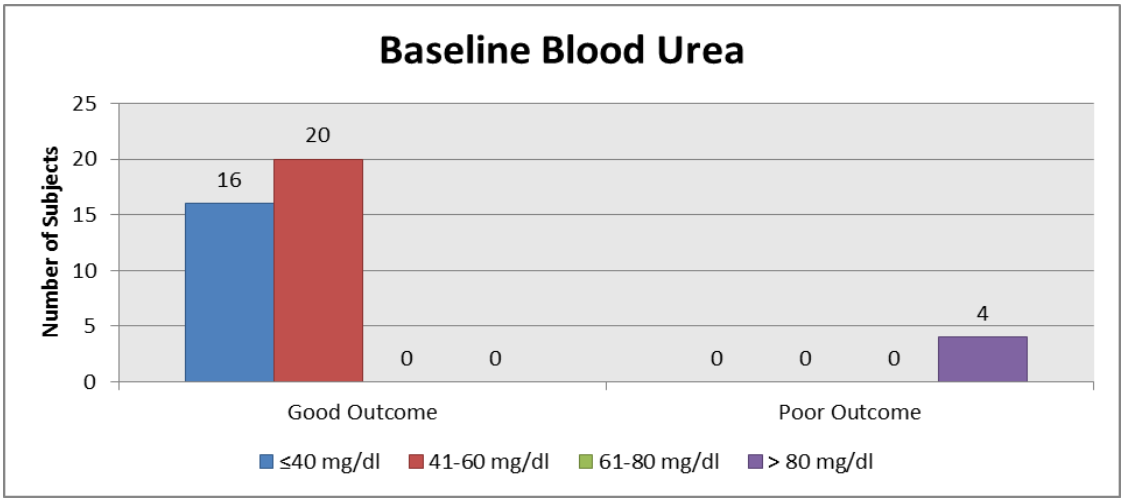



**Figure 11: Baseline Blood Urea**

| Baseline Blood Urea | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **≤40 mg/dl** | 16 | 44.44 | 0 | 0.00 |
| **41-60 mg/dl** | 20 | 55.56 | 0 | 0.00 |
| **61-80 mg/dl** | 0 | 0.00 | 0 | 0.00 |
| **> 80 mg/dl** | 0 | 0.00 | 4 | 100.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Baseline Blood Urea | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 41.41667 | 99.5 |
| **SD** | 8.649938 | 9.036961 |
| **P value Unpaired t test** | | 0.000435* |

By conventional criteria the association between Baseline Blood Urea levels and outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

This indicates that there is a true difference among groups and the difference is significant. In simple terms, when studying the Role of minimally invasive approaches for renal salvagibility in management of Emphysematous Pyelonephritis, the average Blood Urea levels in good outcome (41.42 mg/dl) is predominantly less when compared to bad outcome (99.5 mg/dl). It is statistically significant with a p-value of 0.000435 according to unpaired t test.

**Clinical Significance**

The average Blood Urea levels in good outcome group are meaningfully less than bad outcome by 2.4 times with a mean difference of 58.08 mg/dl. Similarly the prevalence of normal Blood Urea levels are much more in good outcome (44.44%) and prevalence of increased Blood Urea levels is much more in poor outcome (100%)

This difference is true and significant and has not occurred by chance.

**Conclusion**

We conclude that there is meaningfully real increase in risk of developing poor outcomes in persons with increased Blood Urea levels among our study subjects. In short increased Blood Urea levels correlate with poor outcomes in our study subjects.

**Shock at presentation**:

9 out of the total 40 patients (22.5%) presented with shock. The relationship of shock with the outcome was statistically significant (p=0.0014). (Fig 12)

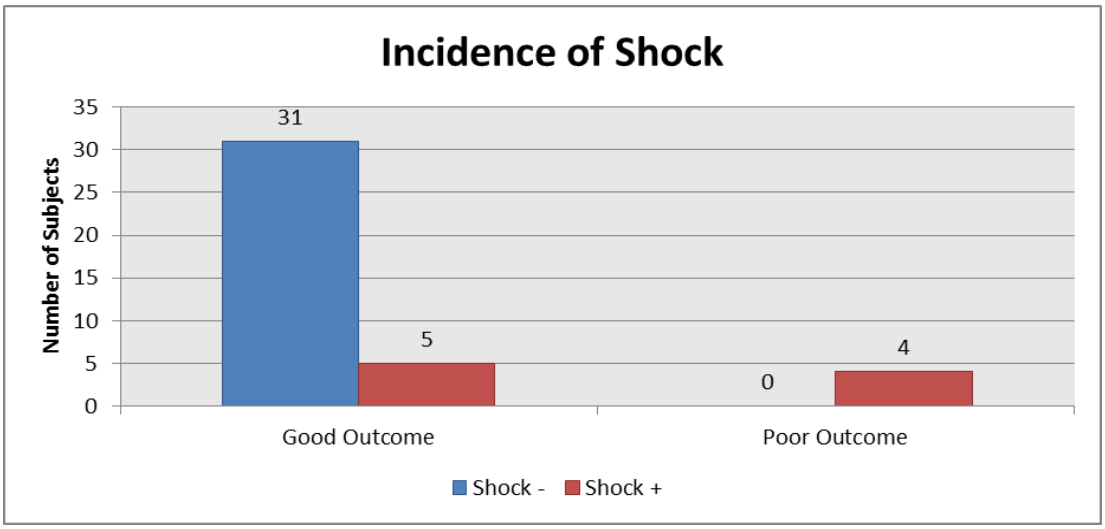



**Figure 12: Shock at presentation**

| Incidence of Shock | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Shock -** | 31 | 86.11 | 0 | 0.00 |
| **Shock +** | 5 | 13.89 | 4 | 100.00 |
| **0** | 0 | 0.00 | 0 | 0.00 |
| **0** | 0 | 0.00 | 0 | 0.00 |
| **0** | 0 | 0.00 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.0014* | |

By conventional criteria the association between Incidence of Shock and Outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of Shock in Emphysematous Pyelonephritis patients is 13.89% in good outcome compared to 100% in bad outcome with a p-value of 0.0014 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of Shock in Emphysematous Pyelonephritis patients was meaningfully 86.11 percentage points less in good outcome compared to bad outcome group
- In our study subjects incidence of Shock leads to 7.2 times increase in occurrence of bad outcomes.

**Conclusion**

We conclude that incidence of Shock is detrimental in nature and can lead to an increasing trend of bad outcomes.

**Mental status on presentation:**

92.5% patients presented in normal mental status while 7.5% had altered mental status on presentation. Out of 3 patients with altered mental status, 3 were in the poor outcome. Thus, altered mental status had a statistically significant relationship with the outcome( p=0.004). (Fig 13)

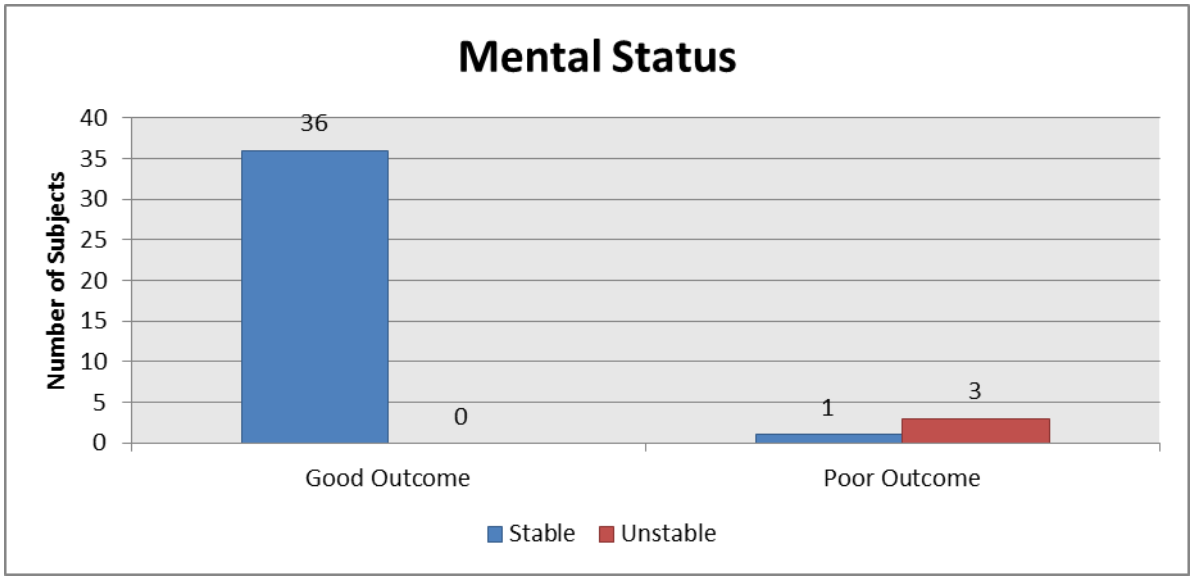



**Figure 13 : Mental status at presentation**

| Mental Status | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Stable** | 36 | 100.00 | 1 | 25.00 |
| **Unstable** | 0 | 0.00 | 3 | 75.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.0004* | |

By conventional criteria the association between mental status and Outcome groups is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of unstable mental status in Emphysematous Pyelonephritis patients is 0% in good outcome group compared to 75% in bad outcome group
- Similarly the incidence of stable mental status in Emphysematous Pyelonephritis patients is 100% in good outcome group compared to 25%

in bad outcome group with a p-value of 0.0004 according to Fisher's Exact test.

- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of stable mental status in Emphysematous Pyelonephritis patients was meaningfully 75.00 percentage points more in good outcome group compared to bad outcome group

In our study subjects incidence of stable mental status leads to 4 times increase in occurrence of good outcomes.

**Conclusion**

We conclude that incidence of unstable mental status is detrimental in nature and can lead to an increasing trend of bad outcomes.

## Blood sugar :

In the present study, the blood sugar value associated with a good outcome was 206.444 +/- 39.151 and the value associated with poor outcome was 375.50+/- 40.553. Blood sugar values at presentation shows significant correlation with the outcome (0.001981) Fig :14.

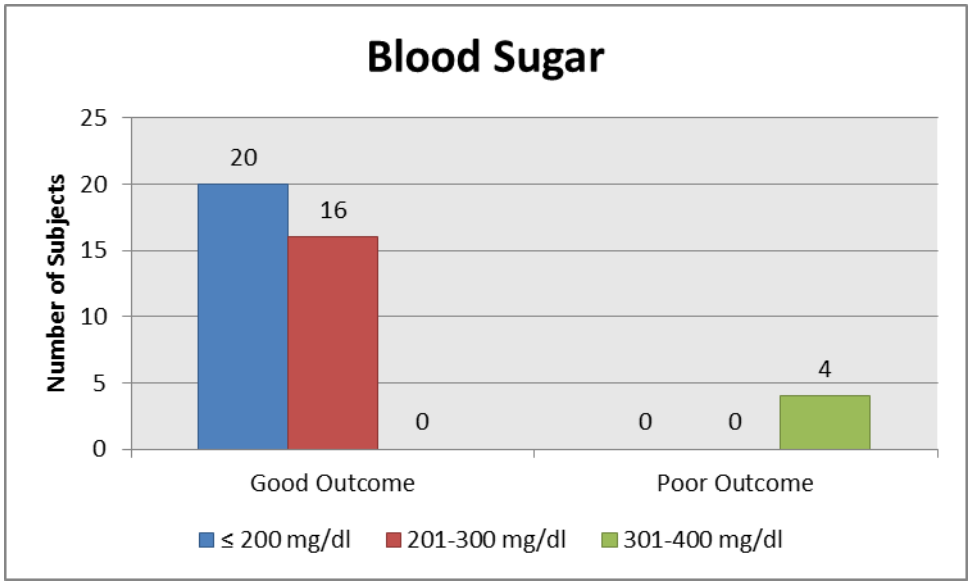



**Figure 14: Blood Sugar**

| Blood Sugar | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **≤ 200 mg/dl** | 20 | 55.56 | 0 | 0.00 |
| **201-300 mg/dl** | 16 | 44.44 | 0 | 0.00 |
| **301-400 mg/dl** | 0 | 0.00 | 4 | 100.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Blood Sugar | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 206.4444 | 375.5 |
| **SD** | 39.15131 | 40.55038 |

**DKA at presentation**:

3 patients (7.5%) were in DKA at presentation. No statistically significant correlation was found between DKA at presentation and outcome (p=0.273) (Fig 15).

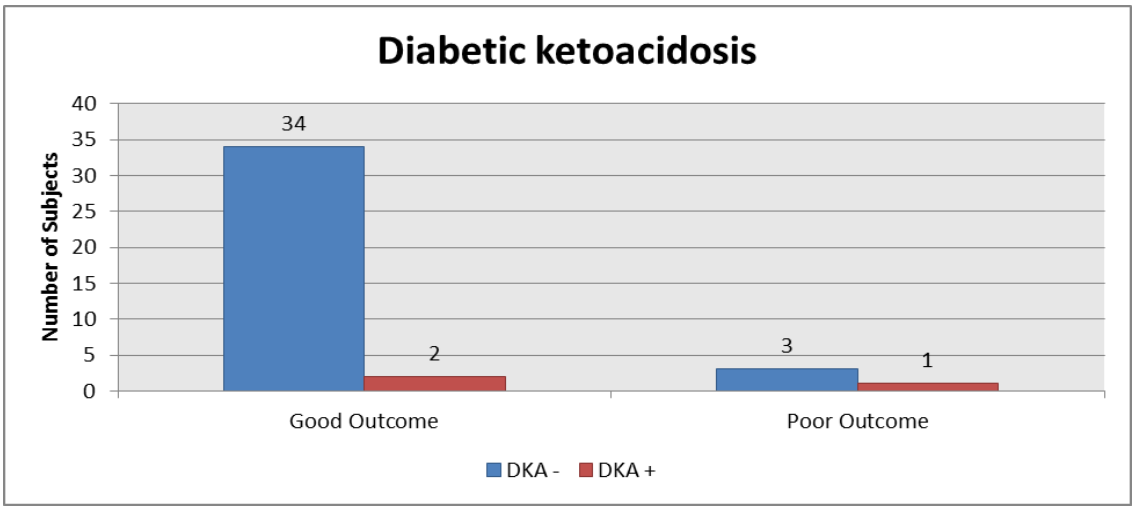



**Figure 15: Diabetic Ketoacidosis**

| Diabetic ketoacidosis | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **DKA -** | 34 | 94.44 | 3 | 75.00 |
| **DKA +** | 2 | 5.56 | 1 | 25.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.2773 | |

**Platelet count:**

In the present study, the correlation between platelet counts and outcome was significant (p=0.0001). The patients were further stratified into two groups based on whether the platelet count was above or below 120000/cu.mm. In the below 120000/cu.mm group, 1/5 were associated with a good outcome and 4/5 were associated with a poor outcome. In the above 120000/cu. mm group, 35/35 were associated with a good outcome and 0/35 were associated with a poor outcome. This reached statistical significance with a p value of 0.0001.Fig: 16

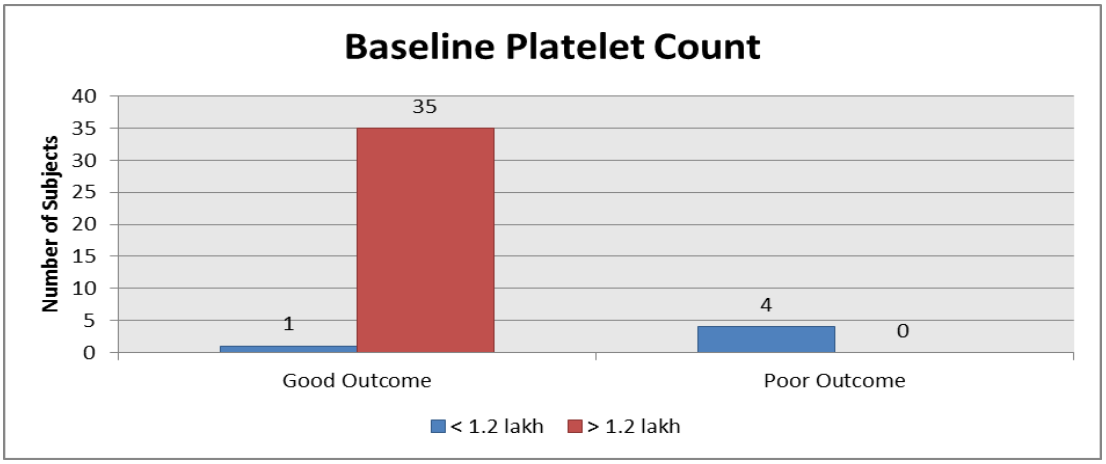



**Figure 16: Platelet Count**

| Baseline Platelet Count | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **< 1.2 lakh** | 1 | 2.78 | 4 | 100.00 |
| **> 1.2 lakh** | 35 | 97.22 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.0001* | |

By conventional criteria the association between Baseline Platelet Count and Outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of baseline platelet count < 1.2 lakh in Emphysematous Pyelonephritis patients is 2.78% in good outcome compared to 100% in bad outcome with a p-value of 0.0001 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of baseline platelet count < 1.2 lakh in Emphysematous Pyelonephritis patients was meaningfully 97.22 percentage points less in good outcome compared to bad outcome
- In our study subjects incidence of baseline platelet count < 1.2 lakh leads to 36 times increase in occurrence of bad outcomes.

**Conclusion**

We conclude that incidence of baseline platelet count < 1.2 lakh is detrimental in nature and can lead to an increasing trend of bad outcomes.

**Total count:**

The correlation between blood TC and outcomes was statistically significant. When patients were further stratified based on whether their total count was above or below 10000/cu.mm. All patients in the below 10000/cu.mm group were associated with a good outcome. In the above 10000/cu.mm group, 8/12 were associated with a good outcome and 4/12 were associated with poor outcome. This association reached statistical significance (p=0.0054) Fig: 17.

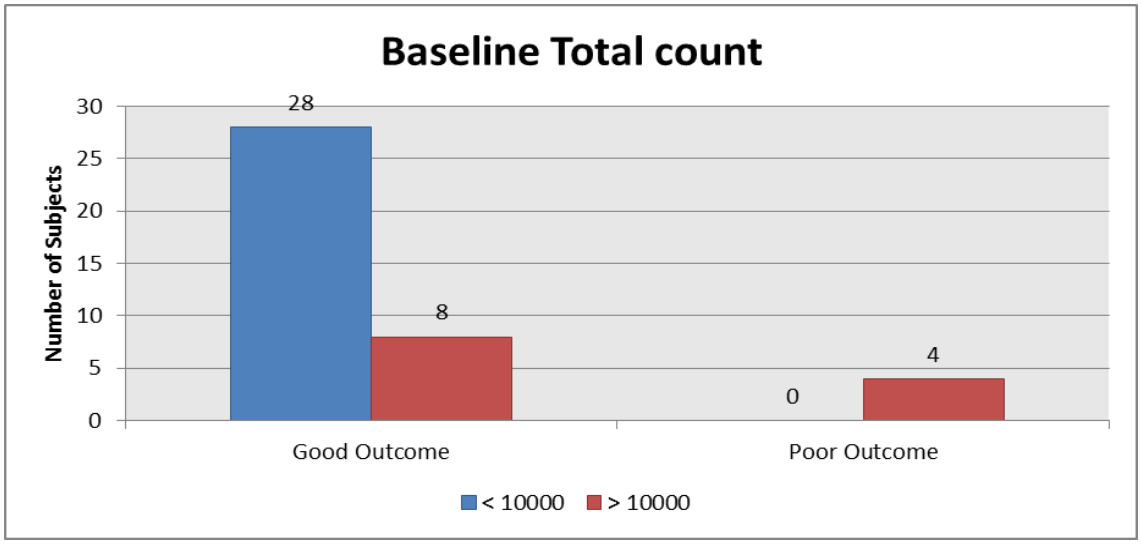



**Figure 17: Total Count**

| Baseline Total count | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **< 10000** | 28 | 77.78 | 0 | 0.00 |
| **> 10000** | 8 | 22.22 | 4 | 100.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.0054* | |

By conventional criteria the association between Baseline Total Count and Outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of baseline Total count > 10000 in Emphysematous Pyelonephritis patients is 22.22% in good outcome compared to 100% in bad outcome with a p-value of 0.0054 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of baseline Total count > 10000 in Emphysematous Pyelonephritis patients was meaningfully 77.78 percentage points less in good outcome compared to bad outcome
- In our study subjects incidence of baseline Total count > 10000 leads to 4.5 times increase in occurrence of bad outcomes.

## Conclusion

We conclude that incidence of baseline Total count > 10000 is detrimental in nature and can lead to an increasing trend of bad outcomes.

## Blood Haemoglobin (Hb):

In the present study, the mean Hb value was 10.75 with a S.D of 0.43. In good outcome patients, the Hb was 10.75 +/- 0.433. In the poor outcome, the Hb was 8.1500 +/- 1.2909. There was no statistically significant correlation between the Hb value and the outcomes since p>0.05 (p=0.0807 Fig :18

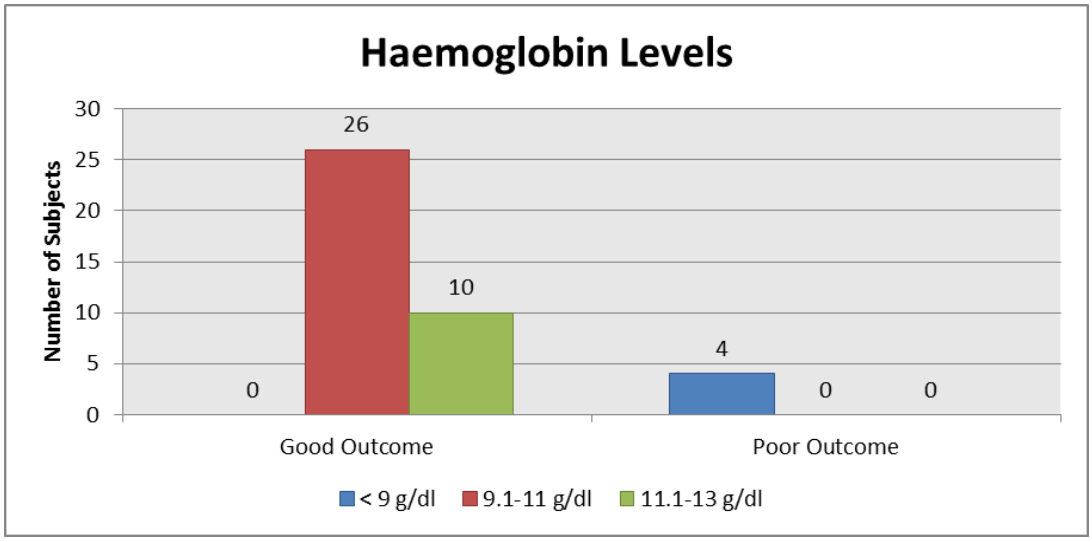



**Figure 18: Haemoglobin levels**

| Haemoglobin Levels | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **< 9 g/dl** | 0 | 0.00 | 2 | 50.00 |
| **9.1-11 g/dl** | 26 | 72.22 | 2 | 50.00 |
| **11.1-13 g/dl** | 10 | 27.78 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |

| Haemoglobin Levels | Good Outcome | Poor Outcome |
|---|---|---|
| **N** | 36 | 4 |
| **Mean** | 10.75 | 9.075 |
| **SD** | 0.433919 | 1.297112 |
| **P value Unpaired t test** | | 0.0807 |

**CT classification**:

The following was the distribution of the patients[8].

Class 1 – 5% (2 patients)

Class 2 – 52.5% (19 patients)

Class 3A- 25% (10 patients)

Class 3B- 15% (6 patients)

Class 4- 7.5% (3 patients) There was correlation made out between CT class and the outcome (p=0.001) (Fig19).

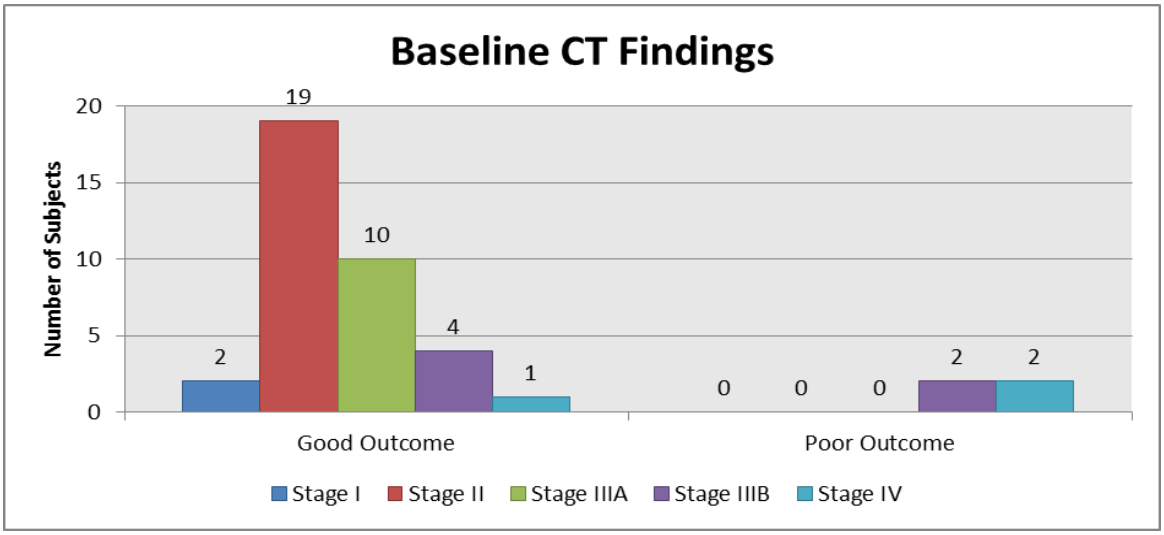



**Figure 19: CT classification**

| Baseline CT Findings | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Stage I** | 2 | 5.56 | 0 | 0.00 |
| **Stage II** | 19 | 52.78 | 0 | 0.00 |
| **Stage IIIA** | 10 | 27.78 | 0 | 0.00 |
| **Stage IIIB** | 4 | 11.11 | 2 | 50.00 |
| **Stage IV** | 1 | 2.78 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.001* | |

By conventional criteria the association between CT findings and Outcome groups is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of CT Stage IV in Emphysematous Pyelonephritis patients is 2.78% in good outcome compared to 50% in bad outcome with a p-value of 0.001 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of CT Stage IV findings in Emphysematous Pyelonephritis patients was meaningfully 47.22 percentage points less in good outcome group compared to bad outcome group
- In our study subjects incidence of CT Stage IV finding leads to 18 times increase in occurrence of bad outcomes.

## Conclusion

We conclude that incidence of CT Stage IV finding in our subjects is detrimental in nature and can lead to an increasing trend of bad outcomes.

**Results of urine culture**:

The commonest organism grown in urine culture was E.coli (75%). E.coli with Proteus was grown in 5%, and other organisms (Klebsiella, Proteus) in 20%.Urine culture result did not correlate with the outcome (p=0.644) (Fig 20).

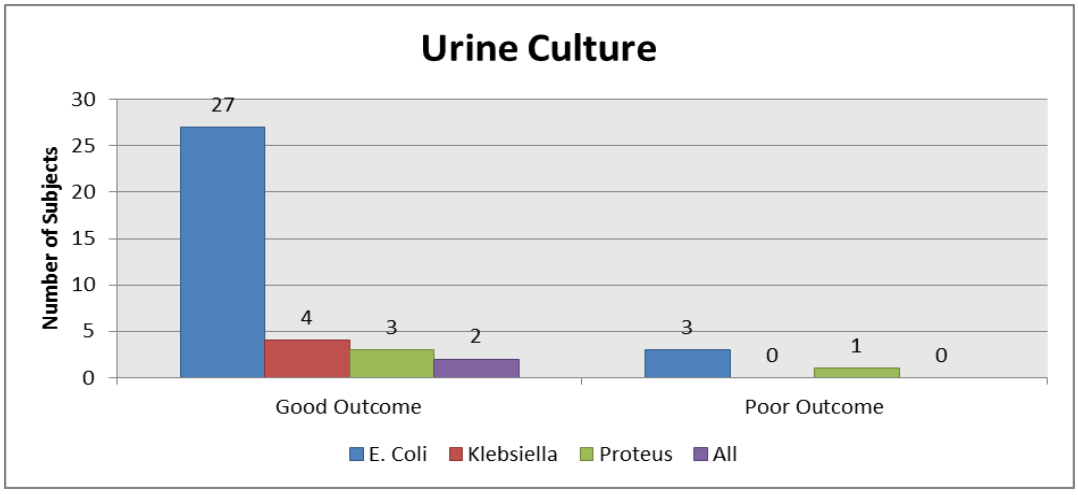



**Figure 20: Urine Culture**

| Urine Culture | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **E. Coli** | 27 | 75.00 | 3 | 75.00 |
| **Klebsiella** | 4 | 11.11 | 0 | 0.00 |
| **Proteus** | 3 | 8.33 | 1 | 25.00 |
| **All** | 2 | 5.56 | 0 | 0.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.6444 | |

**Results of blood culture**:

Blood cultures were positive in 15% of the cases. Of the 6 patients with a positive blood culture, 4 had poor outcome .The relationship between blood culture positivity and outcome reached statistical significance (p=0.0001). (Fig 21)

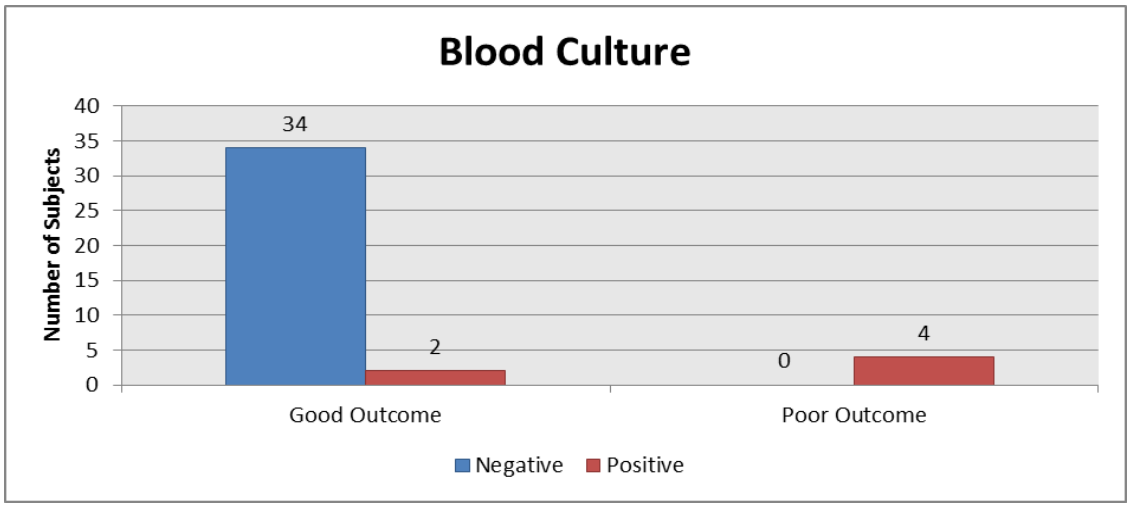



**Figure 21: Blood Culture**

| Blood Culture | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Negative** | 34 | 94.44 | 0 | 0.00 |
| **Positive** | 2 | 5.56 | 4 | 100.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| | **P value Fisher's Exact test** | | 0.0001* | |

By conventional criteria the association between Blood Culture findings and Outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of Blood Culture Positivity in Emphysematous Pyelonephritis patients is 5.56% in good outcome group compared to 100% in bad outcome with a p-value of 0.0001 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of Blood Culture Positivity in Emphysematous Pyelonephritis patients was meaningfully 94.44 percentage points less in good outcome compared to bad outcome
- In our study subjects incidence of Blood Culture Positivity leads to 18 times increase in occurrence of bad outcomes.

**Conclusion**

We conclude that incidence of Blood Culture Positivity finding in our subjects is detrimental in nature and can lead to an increasing trend of bad outcomes.

**Presence of obstruction:**

In the present study, urinary tract obstruction was present in 47.5% of patients. 52.5% patients did not have associated obstruction. Of the 19 patients who had associated urinary tract obstruction, all the 15 were associated with good outcome. Of the 21 patients with no associated obstruction, 100% (21/21) had a good outcome and 0% (0/21) had a poor outcome. (p=0.0001).This implies that presence of obstruction when relieved would assist renal conservation. Fig: 22

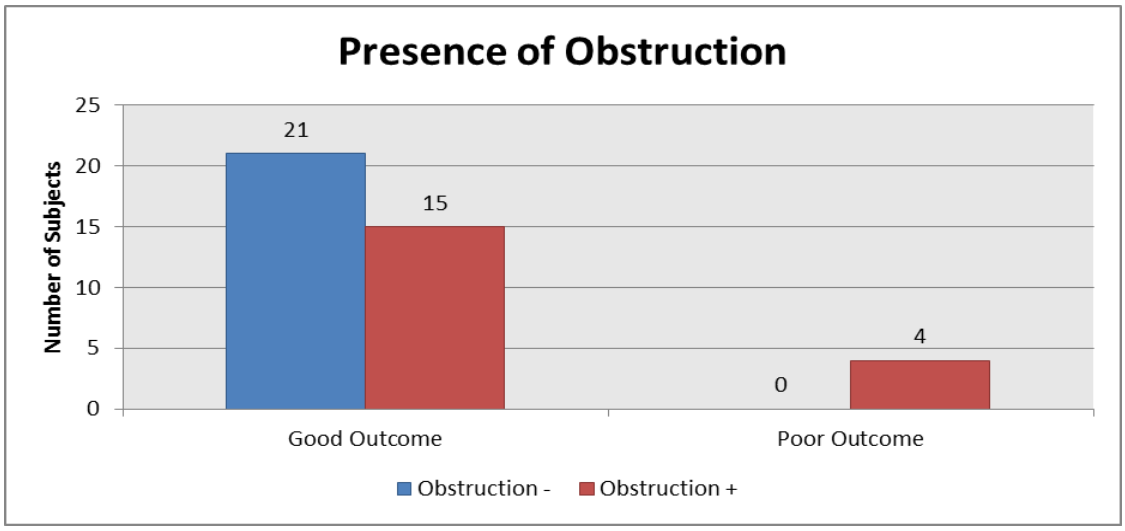



**Figure 22: Presence of Obstruction**

| Presence of Obstruction | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Obstruction -** | 21 | 58.33 | 0 | 0.00 |
| **Obstruction +** | 15 | 41.67 | 4 | 100.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.0001* | |

By conventional criteria the association between Presence of Obstruction and Outcome groups is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- In simple terms, the incidence of Presence of Obstruction in Emphysematous Pyelonephritis patients is 41.67% in good outcome compared to 100% in bad outcome with a p-value of 0.0001 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of Blood Culture Positivity in Emphysematous Pyelonephritis patients was meaningfully 58.33 percentage points less in good outcome compared to bad outcome
- In our study subjects incidence of Blood Culture Positivity leads to 2.4 times increase in occurrence of bad outcomes.

**Conclusion**

We conclude that incidence of Presence of Obstruction finding in our subjects is detrimental in nature and can lead to an increasing trend of bad outcomes.

**Modes of treatment**:

Antibiotics only was used in 10% of patients. DJ stenting was the only modality in 65% of the patients and PCD only in 5%. PCD was combined with DJ stenting in 15% of patients. Open drainage after PCD and DJ stenting failure is 5%. Nephrectomy after PCD failure is 5%. Fig :23

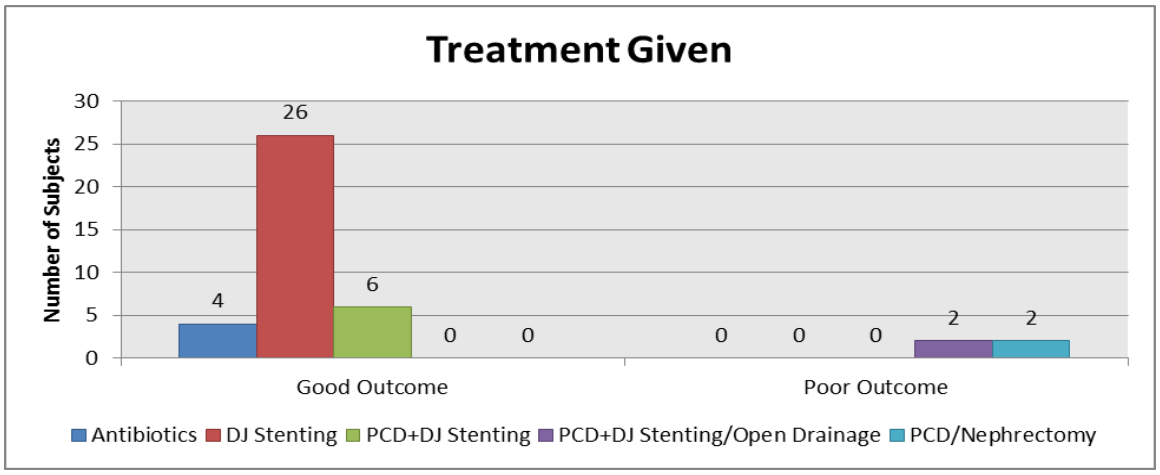



**Figure 23: Modes of Treatment**

| Treatment Given | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Antibiotics** | 4 | 11.11 | 0 | 0.00 |
| **DJ Stenting** | 26 | 72.22 | 0 | 0.00 |
| **PCD+DJ Stenting** | 6 | 16.67 | 0 | 0.00 |
| **PCD+DJ Stenting failed-Open Drainage** | 0 | 0.00 | 2 | 50.00 |
| **PCD failed - Nephrectomy** | 0 | 0.00 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 01774 | |

**Post conservative treatment Platelet count**

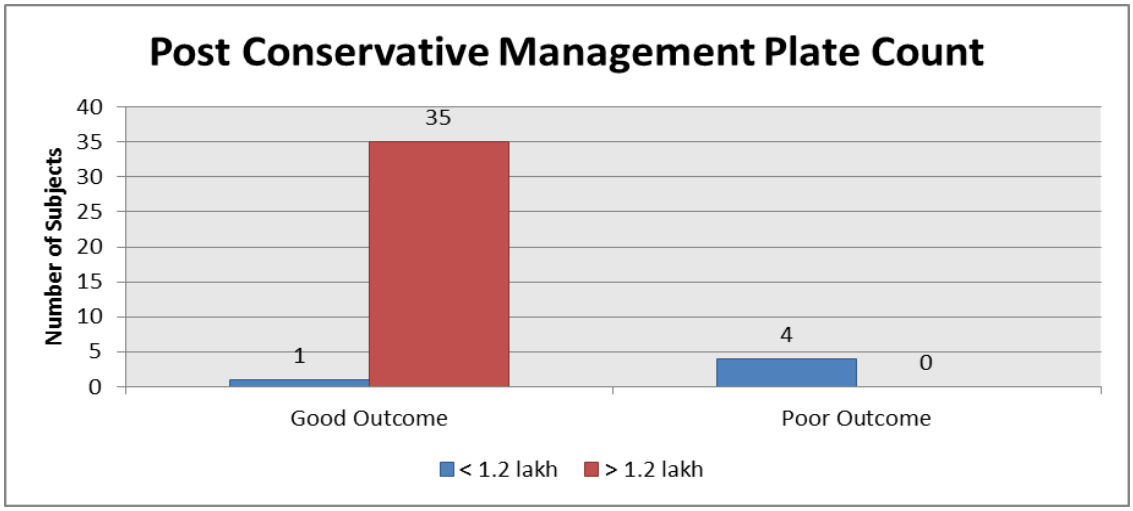



**Figure: 24**

**Post conservative treatment Serum Creatinine**

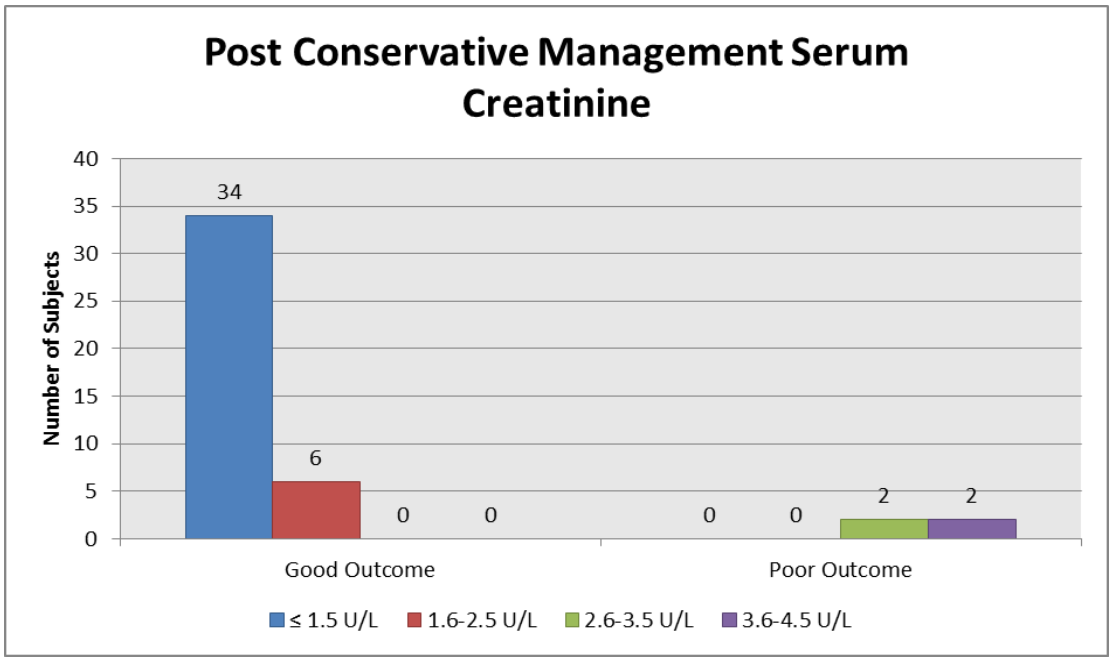



**Figure: 25**

**Post conservative treatment Total count**

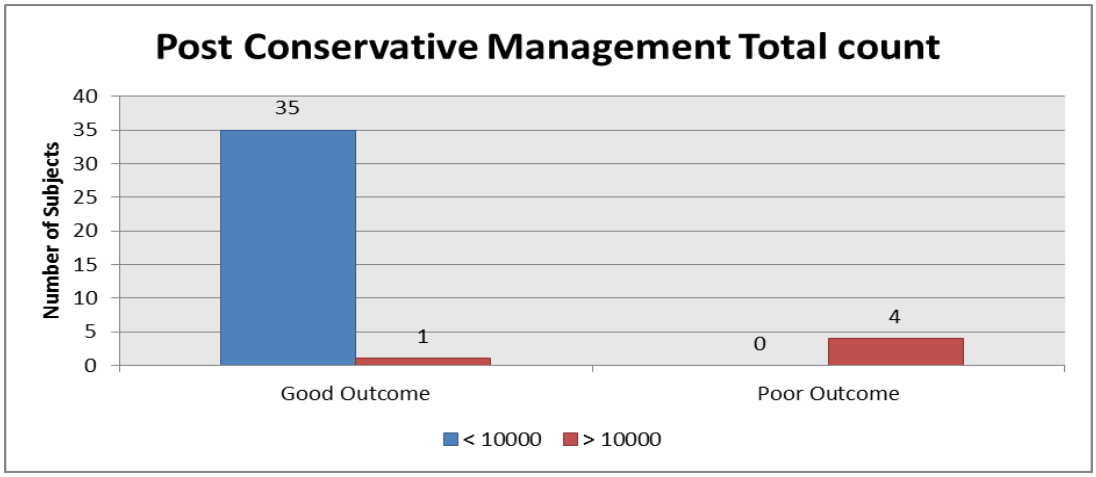



**Figure: 26**

**Risk factors:**

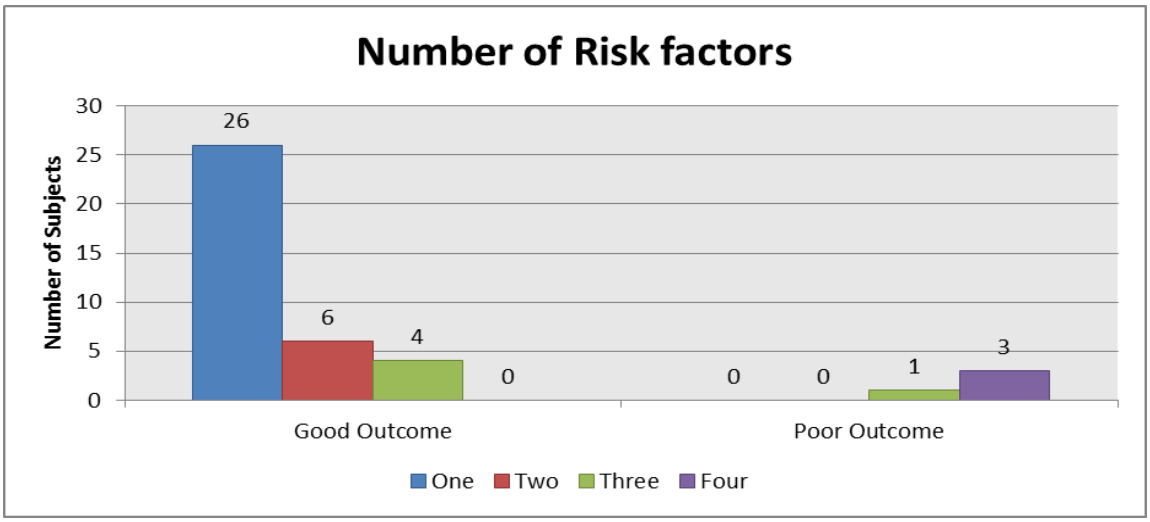



**Figure: 27**

**Type of outcome:**

90% patients (36/40 ) had a good outcome in the form of renal conservation. 10% patients (4/40) had a poor outcome of minimal invasive treatment had renal conservation in form of open drainage - 2/4 and renal loss by nephrectomy - 2/4 (Fig 28).

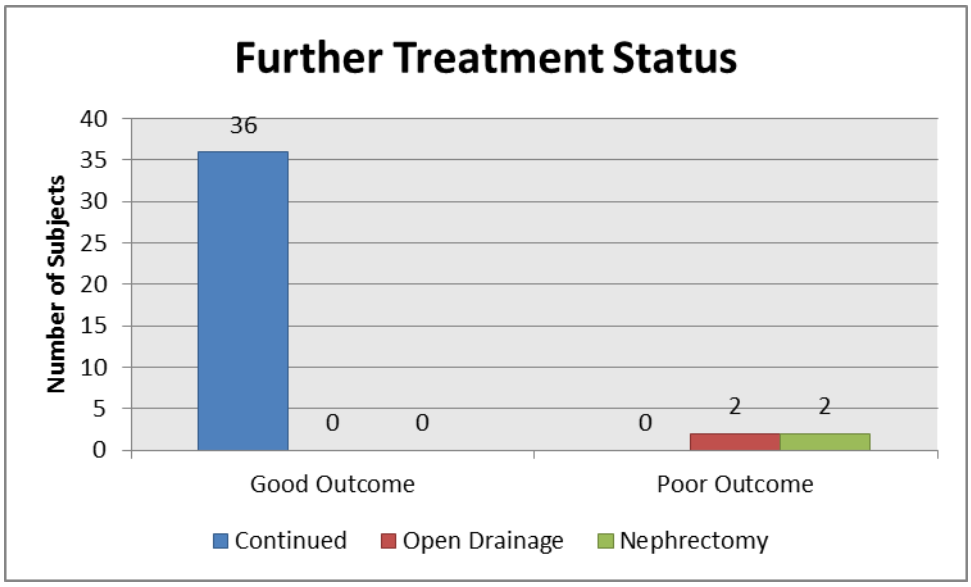



**Figure 28: Type of Outcome**

| Further Treatment | Good Outcome | % | Poor Outcome | % |
|---|---|---|---|---|
| **Continued** | 36 | 100.00 | 0 | 0.00 |
| **Open Drainage** | 0 | 0.00 | 2 | 50.00 |
| **Nephrectomy** | 0 | 0.00 | 2 | 50.00 |
| **Total** | 36 | 100 | 4 | 100.00 |
| **P value Fisher's Exact test** | | | 0.0001* | |

By conventional criteria the association between Treatment Given and Outcome is considered to be statistically significant since $p < 0.05$.

**Statistical Significance**

- The incidence of minimally invasive treatment given in Emphysematous Pyelonephritis patients is 100% in good outcome compared to 0% in bad outcome .
- The incidence of invasive treatment given in Emphysematous Pyelonephritis patients is 0% in good outcome compared to 100% in bad outcome group with a p-value of 0.0001 according to Fisher's Exact test.
- This indicates that there is a true difference among the study groups and the difference is significant and has not occurred by chance.

**Clinical Significance**

- The incidence of minimally invasive treatment given in Emphysematous Pyelonephritis patients was meaningfully 100 percentage points less in good outcome compared to bad outcome
- In our study subjects incidence of minimally invasive treatment given leads to 100 times increase in occurrence of bad outcomes.

**Conclusion:**

We conclude that incidence of minimally invasive treatment given correlates with an increasing trend of good outcomes.

# DISCUSSION

## DISCUSSION

40 patients were included prospectively during the study period. Comparison made with the results of the present study with other studies.

| Sl. No | Study | Design | Sample | Finding |
|---|---|---|---|---|
| 1. | Huang et al8 | Prospective | 48 | Class 1 or 2 & class 3 or 4 with <2 risk factors-conservation Others-nephrectomy |
| 2. | Michaeli et al6 | Retrospective | 54 | Resuscitation, early antibiotics, relief of obstruction & early nephrectomy. |
| 3. | Shokeir et al12 | Retrospective | 20 | Nephrectomy |
| 4. | Bum Soo Park Et al5 | Retrospective | 17 | Conservation in selected cases |
| 5. | Chen et al23 | Retrospective | 25 | Antibiotics with CT guided drainage |
| 6. | Wan et al 19 | Retrospective | 38 | Predictors of high risk – S.Creatinine & Platelet count |
| 7. | Present study | prospective | 40 | Minimal invasive surgeries feasible. Predictors of poor outcome identified. |

**Fig 29: Comparative study**

**Age**: Mean age in the present study is 55.12 yrs which is comparable with other studies [3,5, 6, 7, 8, 12 .]

**Sex distribution**: In our study, there was a female predominance, which is seen in other studies also.

**Side of involvement**: In the present study, there was a predominance of left over the right side. In other studies also, a similar female predominance is seen.

**Presenting complaints:** The predominant mode of presentation in the present study was fever associated with loin pain. This is similar to other studies.[8,12.]

**Duration of symptoms before presentation:**

The mean duration of symptoms before presentation was 11.52 days in our study. In Chen et al's study [3], it was 18 +/- 8.64 days[3]. In our study, the mean duration of symptoms before presentation in the good outcome was 11.52 days and in the poor outcome was 6.25 days. In comparison, in Huang et al's study, the duration of symptoms prior to diagnosis in the good outcome was 8.2 days and in the poor outcome was 6.1 days [8.]

**Presence of Diabetes mellitus**:

DM was present in 87.5% of patients in our study which correlates well with the studies of Chen et al [3] and Shokeir et al [12].The prevalence of DM in Huang et al's study was 96% [8].

**Presence of shock**:

In the present study,[5/8] (100%) of the patients in poor outcome presented with shock and (13.89%) of the patients in good outcome presented with shock. In comparison, in Huang et al's study 56% in the poor outcome and 17% of patients in the good outcome presented with shock [8].

**Altered mental status at presentation**:

In the present study, [3/8] (75.0%) of the patients in poor outcome presented with altered mental status and 0/36 (0%) of the patients in good outcome presented with altered mental status. In comparison, in Huang et al's study 50% in the poor outcome and 3% of patients in the good outcome presented with altered mental status[8].

**Altered renal parameters**:

In the present study, the mean serum creatinine in patients with good outcome was 1.44+/-0.585. The mean serum creatinine in patients with poor outcome was3.625+/- 0.485.This reached statistical significance (p=0.040). Then the patients were stratified based on a cut off of serum creatinine (1.5mg/dl) and patients analysed with regards to the outcome. In the <1.5mg/dl group, 20/20 patients fell under the good outcome and 0/20 patients fell under the poor outcome. In the >1.5mg/dl group, 16/20 fell under the good outcome and 4/20 patients fell under the poor outcome . This was statistically significant (p=0.001092).

**Management and outcome according to radiological classes:**

In the present study, 100% of patients in class 1(2/2) had a good outcome which is comparable with the Huang et al study[8].In class 2, 100% patients(19/19) had a good outcome. This is comparable with the Huang et al study[8]. In class 3A, 100% patients (10/10) had a good outcome and 0% patients (0/10) had a poor outcome. In comparison, in the Huang et al study[8], there was a 100% good outcome. In class 3B, 0% patients (4/6) had a good outcome and 33.33% patients(2/6) had a poor outcome. In comparison, in the Huang et al study[8], there was a 49% poor outcome. In class 4, 0% patients (1/3) had a good outcome and 66.66% patients(2/3) had a poor outcome. In comparison, in the Huang et al study[8], there was a 75% poor outcome.

**Management and outcome**

In the present study, use of antibiotics only was associated with a good outcome in 100% and a poor outcome in 0% patients, while in Huang et al's study, it was associated in 60% and 0% with good and poor outcomes respectively[8]. The use of PCD only was associated with a good outcome in 0% and a poor outcome in 100% patients, while in Huang et al's study, it was associated in 66% and 20% with good and poor outcomes respectively[8]. In the present study, PCD with DJ stenting was associated with a good outcome in 75% and a poor outcome in 25% patients. In patients treated with DJ stenting only, there was a 100% successful outcome.

**Urine cultures:**

In the present study, E.coli was grown in 75% of patients and combined in 5% and either proteus/Klebsiella pneumoniae in 20% of patients. In comparison, in the study of Bum Soo Park et al, 52% grew E.coli and 24% grew Klebsiella pneumoniae. In their study, 24% did not show any growth in the urine[5].

**Blood cultures:**

In the present study, blood cultures were positive in 15% of patients. This compares well with Wan et al's study[7] in which 42.10% had positive blood cultures but is much less than in Bum Soo Park et al's study5 in which 59% had positive blood cultures.

**Obstruction:**

In the present study, obstruction was present in 47.5% of patients. In this group, when obstruction was relieved, there was a 78% association with good outcome. In the good outcome, 41.75% patients had associated obstruction. This contrasts with Huang et al's study in which good outcome was associated with obstruction in 25% patients only[8].

**Platelet count:**

In the present study, 2.78% of the patients in the good outcome and 100% patients in the poor outcome had thrombocytopenia (platelet count < 120000). This is comparable to the study by Huang et al[8], in which, 28% in the

good outcome and 81% in the poor outcome were associated with a platelet count of < 120000. This relationship reached statistical significance in both the present and Huang et al's study[8].

**Total count:**

In the present study, leucocytosis in good outcome group was 22.22% and in the poor outcome group was 100%. In comparison, in the study by Wan et al[7], in both it reached clinical significance

.

# SUMMARY

# SUMMARY

Of the total 40 patients included in the study the following were the findings.

- Incidence of Emphysematous pyelonephritis is more in the females.
- Left side predominates over the right side in incurrence.

- The most common presenting features were fever and loin pain.
- Emphysematous pyelonephritis commonly affects the diabetics.
- Patients can have unrelated clinical features and present in the emergency casualty

- CT KUB is more useful in diagnosis than USG and Xray KUB.
- Urine culture is predominantly positive – the most common organism being *E.coli spp*.

- The organisms grown in blood culture are the same as in urine cultures.
- Relief of the obstruction assists renal conservation, when there is an associated urinary tract obstruction

- Various modalities of treatment like antibiotics, PCD, DJ stenting either alone or in combination make minimal invasive approaches possible in renal salvagibility feasible in 95% of patients.

- Clinical factors like shock or altered sensorium at presentation, presense or absence of associated urinary tract obstruction, laboratory parameters like raised serum creatinine, raised TC, positive blood cultures, reduced platelet counts are all significant factors in determining the outcome during attempted renal conservation out of minimal invasive management.

# CONCLUSION

# CONCLUSIONS

1. Minimal invasive management for renal conservation has a definite role in properly selected patients of emphysematous pyelonephritis.
2. The predicting factors at presentation can alter the conservatively managed cases of emphysematous pyelonephritis lean towards nephrectomy.

- Shock
- Altered mental status
- Raised serum creatinine
- Total count >10000/cmm
- Platelet count < 120000/cmm
- Positive blood cultures
- Absence of urinary tract obstruction.

# PHOTOGRAPHS

# PHOTOGRAPHS

**Fig 30: Xray KUB showing gas in RT renal area**

**Fig 31: Xray KUB showing gas in Lt renal area**

**Fig 32: USG KUB – dirty white shadow**

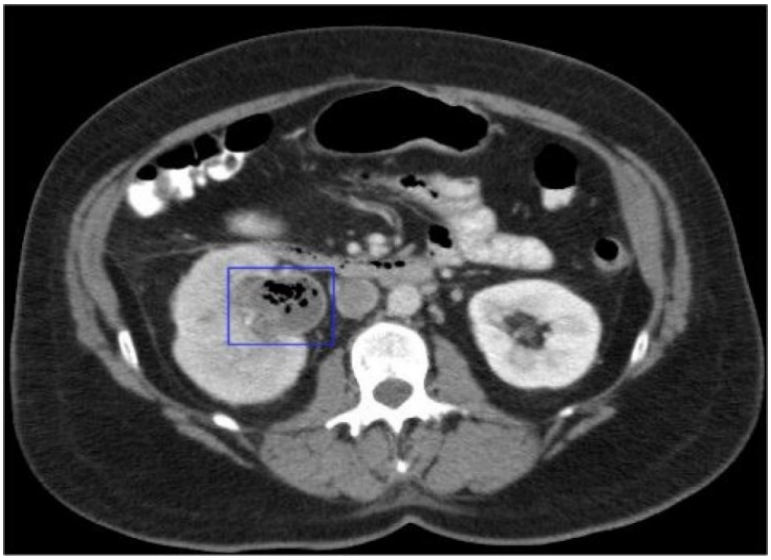


**Fig 33: CT KUB Class 1 EPN**

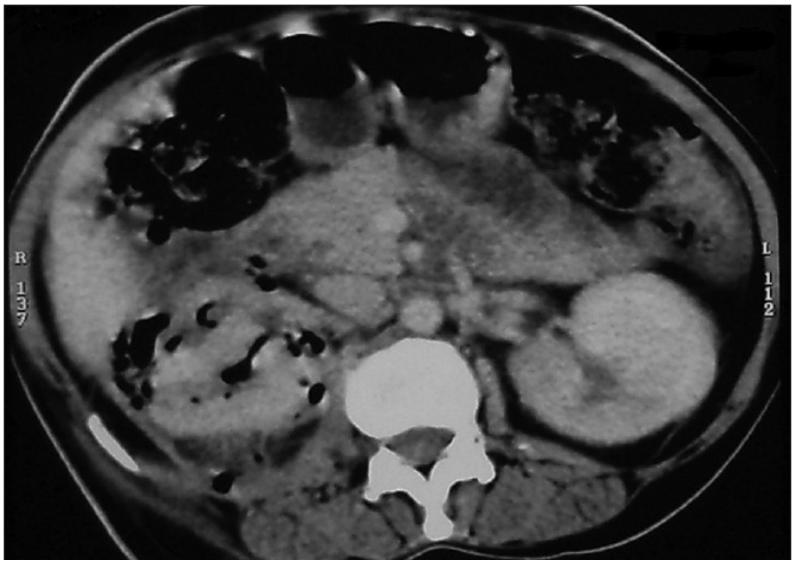



**Fig 34: CT KUB Class 3B EPN**

**Fig 35: CT KUB Class 3A EPN**

**Fig 36: CT KUB - class IV EPN**

**FIG 37: CT KUB – CLASS III B EPN**

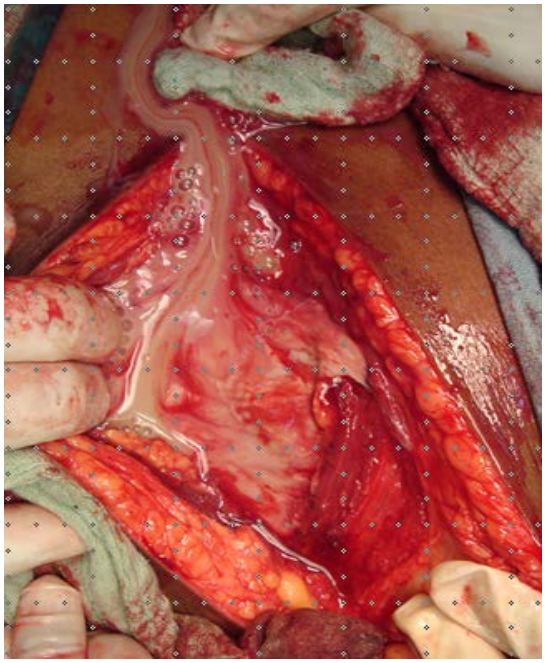


**Fig 38: Nephrectomy in progress**

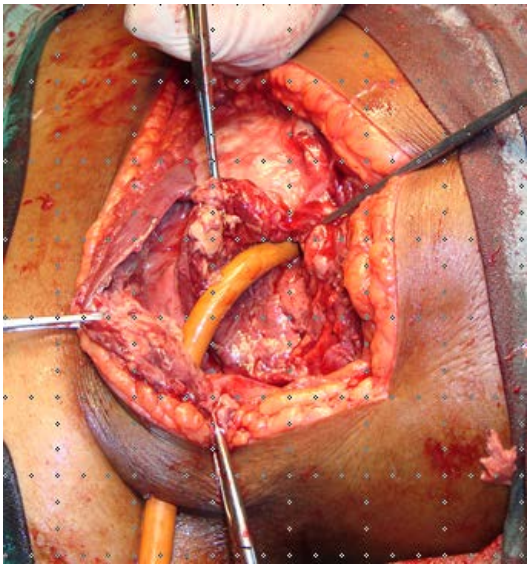


**Fig 39: Open drainage of RT kidney EPN**

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## BIBLIOGRAPHY


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# ANNEXURES

**ANNEXURE I**

**INSTITUTIONAL ETHICAL COMMITTEE**
**GOVT.KILPAUK MEDICAL COLLEGE,**
**CHENNAI-10**
**Protocol ID No.17/11/2014 Dt: 20 .01.2015**
**CERTIFICATE OF APPROVAL**

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Prospective Study of Role of minimally invasive approaches for renal salvagibility in management of Emphysematous Pyelonephritis" submitted by Dr.Saraswathi.S, MCh Urology, PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

20/1/15

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

19/1/2015

GOVT. KILPAUK MEDICAL COLLEGE
20 JAN 2015
Kilpauk, Chennai-10.

6 Comp.> ME 1> Ethical Committee

**Annexure 2**

**PROFORMA**

Name Date of admission

IP no Date of discharge

Age

Sex

Complaints

Duration of symptoms

H/O DM (along with duration and treatment)

Clinical examination

Level of consciousness Shock / BP

Loin Tenderness Temp:

Investigations at presentation

Blood sugar

Serum creatinine & Blood urea

Total WBC count & Haemoglobin

Platelet count

Urine acetone

Urine culture & sensitivity

Blood culture

Mode of diagnosis

CT class

Presence of obstruction

Treatment category

Antibiotics used

Post conservative management serum creatinine

Post conservative management platelet count

Post conservative management recovery of signs and symptoms

Post conservative management CT

Follow up treatment if any

Outcome

Number of days of hospital stay

**ANNEXURE 3**

# சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு:

சிறுநீரக அறுவை சிகிச்சை பிரிவு

கீழ்ப்பாக்கம் மருத்துவக் கல்லூரி

பங்கு பெறுபவரின் பெயர்

பங்கு பெறுபவரின் எண்

பங்கு பெறுபவர் இதனை (√) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன். ☐

நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்தும் கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறோன். ☐

இந்த ஆய்வில் ஒருமுறை 5 மி இரத்தம் பரிசோதனைக்காக எடுத்துக் கொள்ளப்படும் என்பதை அறிவேன். ☐

பங்கேற்பவரின் கையொப்பம்_____இடம் _____

தேதி இடம் _____தேதி_____

பங்கேற்பவரின் பெயர் மற்றும் விலசம்
சாட்சியாளரின் கையொப்பம்

இடம் _____ தேதி _____
சாட்சியாளரின் பெயர் மற்றும் விலாசம்

ஆய்வாளரின் கையொப்பம்
இடம் _____ தேதி _____

ஆய்வாளரின் பெயர் _____

# ANNEXURE IV

# MASTER SHEET I

| SI. No | Name | IP.NO | Age Yrs | Sex F/ M | Side LT/ RT/ B/L | DM D/ND Know/ NK | Modality of DM treatment | Complaints LP+F/L/ ALT. SENSORIUM | C/E LT/ AM/ AD | Sr, Creatinine mg/ dl | Blood Urea mg/dl | Shock +/_ | Mental Status S/US | Blood Sugar mg/dl | Platelet Count- < 1.2 lakh >1.2 | Total Count - cells <10000 >10000 | HB gm% | DKA +/_ | Urine C/S E.C /K/ P | Blood C/S P/N |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Koteeswari | 14132 | 54 | F | LT | DM | R/OHA | LP+F | LT | 2.0 | 41 | + | S | 190 | >1.2 | <10000 | 9.7 | _ | E.C | N |
| 2 | Velu | 14321 | 56 | M | LT | DM | R/OHA | LP+F | LT | 2.0 | 42 | + | S | 200 | >1.2 | >10000 | 10.0 | _ | E.C | N |
| 3 | Meenambal | 14501 | 53 | F | LT | DM | R/OHA | LP+F | LT | 1.7 | 45 | _ | S | 250 | >1.2 | <10000 | 10.1 | _ | E.C | N |
| 4 | Unnamalai | 14637 | 57 | M | LT | DM | R/OHA | LP | LT | N | 28 | _ | S | 198 | >1.2 | <10000 | 10.6 | _ | E.C | N |
| 5 | Selvaraj | 14718 | 58 | M | LT | DM | R/OHA | LP+F | AM | 1.6 | 60 | + | S | 210 | >1.2 | >10000 | 10.5 | _ | K | N |
| 6 | Jeenath | 4600 | 52 | F | LT | DM | R/INSUL | LP+F | LT | 1.8 | 52 | _ | S | 199 | >1.2 | <10000 | 9.9 | _ | E.C | N |
| 7 | Kanchi | 5752 | 39 | F | LT | D – R/D | - | LP | LT | N | 29 | _ | S | 170 | >1.2 | <10000 | 10.7 | _ | E.C | N |
| 8 | Sundharam | 6978 | 78 | M | LT | DM | R/OHA | LP+F | AM | 3.9 | 89 | + | US | 315 | <1.2 | >10000 | 8.2 | _ | E.C | P |
| 9 | Suseela | 9456 | 71 | F | LT | DM | R/OHA | LP+F | LT | N | 42 | _ | S | 210 | >1.2 | <10000 | 10.9 | _ | K | N |
| 10 | Saroja | 1032 | 50 | F | LT | DM | R/OHA | LP | LT | N | 35 | _ | S | 183 | >1.2 | <10000 | 11.0 | _ | E.C | N |
| 11 | Rani | 2215 | 53 | F | LT | DM | R/OHA | LP+F | LT | N | 39 | _ | S | 205 | >1.2 | <10000 | 11.1 | _ | E+ K/P | P |
| 12 | Savithri | 9942 | 49 | F | LT | D - R/D | - | LP | LT | N | 30 | _ | S | 186 | >1.2 | <10000 | 11.1 | _ | E.C | N |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 13 | Kathayee | 10141 | 61 | F | B/L | DM | R/OHA | ALT.SE NS | AD | 3.5 | 100 | + | US | 397 | <1.2 | >10000 | 8.0 | _ | P | P |
| 14 | Devi | 10964 | 47 | F | LT | DM | R/OHA | LP+F | LT | N | 32 | _ | S | 207 | >1.2 | <10000 | 10.6 | _ | E.C | N |
| 15 | Roselin | 11630 | 63 | F | LT | DM | R/OHA | LP+F | AM | 1.9 | 48 | + | S | 230 | >1.2 | <10000 | 10.7 | _ | E.C | N |
| 16 | Mangayarkarasi | 12018 | 46 | F | LT | DM | R/OHA | LP | LT | N | 35 | _ | S | 193 | >1.2 | <10000 | 10.8 | _ | E+ K/P | N |
| 17 | Mohan | 12999 | 64 | M | LT | DM | R/INS | LP+F | LT | N | 40 | _ | S | 200 | >1.2 | <10000 | 10.9 | _ | E.C | N |
| 18 | Powlin | 13567 | 45 | F | LT | D – R/D | - | LP | LT | N | 29 | _ | S | 163 | >1.2 | <10000 | 10.4 | _ | P | N |
| 19 | Suseela devi | 14748 | 65 | F | B/L | DM | R/OHA | ALT.SE NS | AD | 4.1 | 98 | + | US | 400 | <1.2 | >10000 | 8.3 | + | E.C | P |
| 20 | Kanagaselvi | 14835 | 63 | F | LT | DM | R/OHA | LP+F | AM | 3.0 | 111 | + | S | 390 | <1.2 | >10000 | 8.1 | _ | E.C | P |
| 21 | Rajashekar | 15117 | 47 | M | LT | DM | R/OHA | LP+F | LT | N | 32 | _ | S | 21 | >1.2 | <10000 | 10.3 | _ | K | N |
| 22 | Kuppusamy | 15256 | 48 | M | RT | DM | R/OHA | LP+F | LT | N | 36 | _ | S | 22 | >1.2 | <10000 | 10.1 | _ | E.C | N |
| 23 | Raja Manikkam | 1076 | 62 | M | B/L | DM | IRREG | LP+F | AD | 2.1 | 40 | + | S | 23 | <1.2 | >10000 | 10.2 | + | E.C | P |
| 24 | Muthupandi | 2089 | 55 | M | RT | DM | R/OHA | LP+F | LT | 1.6 | 42 | _ | S | 24 | >1.2 | <10000 | 10.5 | _ | P | N |
| 25 | Pandiselvan | 2951 | 51 | M | RT | ND | - | LP | LT | N | 40 | _ | S | 25 | >1.2 | <10000 | 10.9 | _ | E.C | N |
| 26 | Rajeshwari | 3398 | 59 | F | RT | ND | - | LP+F | LT | 1.7 | 50 | _ | S | 26 | >1.2 | <10000 | 11 | _ | K | N |
| 27 | Alagu Meenatchi | 4136 | 55 | F | RT | ND | - | LP+F | LT | N | 42 | _ | S | 27 | >1.2 | <10000 | 11.2 | _ | E.C | N |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 28 | Karuppayee | 5839 | 44 | F | RT | ND | - | LP+F | LT | N | 47 | _ | S | 28 | >1.2 | <10000 | 11.3 | _ | E.C | N |
| 29 | Rakkammal | 6130 | 66 | F | RT | ND | - | LP+F | LT | N | 45 | _ | S | 29 | >1.2 | <10000 | 11.4 | _ | P | N |
| 30 | Selvi | 6956 | 43 | F | RT | DM | R/OHA | LP | LT | N | 43 | _ | S | 30 | >1.2 | <10000 | 11.0 | _ | E.C | N |
| 31 | Sundharambal | 7177 | 56 | F | RT | DM | R/OHA | LP+F | LT | 1.6 | 55 | _ | S | 31 | >1.2 | <10000 | 11.2 | _ | E.C | N |
| 32 | Soundharam | 8570 | 54 | F | RT | DM | R/OHA | LP | LT | N | 41 | _ | S | 32 | >1.2 | <10000 | 11.3 | _ | E.C | N |
| 33 | Lakshmi | 9003 | 57 | F | RT | DM | R/OHA | LP+F | AM | 1.7 | 49 | _ | S | 33 | >1.2 | >10000 | 11.1 | _ | E.C | N |
| 34 | Poochi | 10881 | 53 | F | RT | DM | R/OHA | LP | LT | N | 32 | _ | S | 34 | >1.2 | <10000 | 11.0 | _ | E.C | N |
| 35 | Kaaliamal | 10993 | 59 | F | RT | DM | R/OHA | LP+F | AM | 1.8 | 52 | _ | S | 35 | >1.2 | >10000 | 10.9 | _ | E.C | N |
| 36 | Chellamal | 11490 | 51 | F | RT | DM | R/OHA | LP | LT | N | 28 | _ | S | 36 | >1.2 | <10000 | 10.6 | _ | E.C | N |
| 37 | Muthammal | 12734 | 65 | F | RT | DM | R/OHA | LP+F | LT | 2.0 | 55 | _ | S | 37 | >1.2 | >10000 | 10.7 | + | E.C | N |
| 38 | Saratha | 13561 | 64 | F | RT | DM | R/OHA | LP | LT | N | 33 | _ | S | 38 | >1.2 | <10000 | 11.0 | _ | E.C | N |
| 39 | Mangalam | 14587 | 68 | F | LT | DM | R/OHA | LP+F | LT | 1.8 | 50 | _ | S | 39 | >1.2 | >10000 | 11.1 | _ | E.C | N |
| 40 | Parvathi | 14920 | 67 | F | LT | DM | IRREG | LP+F | AM | 1.9 | 52 | + | S | 40 | >1.2 | >10000 | 11.2 | _ | E.C | N |

# MASTER SHEET II

| S.no | Name | Age Yrs | Sex F/M | IP.NO | CT I/II/ III/IV | No of Risk factor | Treatment Details Ab/DJ/PCD +DJ | Post op platelet level - lakhs | Post op Sr. creatinine & Total count | Post op symptom and sign recovery/days | Post op CT | Further treatment | Outcome G/MI G/I Poor/Invas |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Koteeswari | 54 | F | 14132 | IIIA | 2 | DJ STENTING | 1.7 | 1.2/9500 | Good/ 3days | Gas resolved | continue | Good/MI |
| 2 | Velu | 56 | M | 14321 | IIIA | 3 | DJ STENTING | 1.9 | 1.1/9500 | Good/3 days | No HUN | continue | Good/MI |
| 3 | Meenambal | 53 | F | 14501 | IIIA | 1 | DJ STENTING | 2.0 | 1.1/9666 | Good/2 days | Gas resolved | continue | Good/MI |
| 4 | Unnamalai | 57 | M | 14637 | II | _ | DJ STENTING | 2.7 | 1.0/9000 | Good/ 1day | Gas resolved | continue | Good/MI |
| 5 | Selvaraj | 58 | M | 14718 | IIIB | 3 | PCD+DJ STENTING | 2.2 | 1.4/9119 | Good/3 days | No HUN | continue | Good/MI |
| 6 | Jeenath | 52 | F | 4600 | II | 1 | DJ STENTING | 2.5 | 1.2/8350 | Good/2 days | Gas resolved | continue | Good/MI |
| 7 | Kanchi | 39 | F | 5752 | I | _ | DJ STENTING | 3.2 | 1.0/8600 | Good/ 1day | Gas resolved | continue | Good/MI |
| 8 | Sundharam | 78 | M | 6978 | IIIB | 4 | PCD+DJ STENTING | 1.1 | 3.0/12000 | Poor/3days | Mass+ | Open drainage | Good / invasive |
| 9 | Suseela | 71 | F | 9456 | II | _ | DJ STENTING | 3.0 | 1.0/8500 | Good/ 2 days | Gas resolved | continue | Good/MI |
| 10 | Saroja | 50 | F | 1032 | I | _ | ANTIBIOTIC | 2.8 | 0.8/8200 | Good/2 days | Gas resolved | continue | Good/MI |
| 11 | Rani | 53 | F | 2215 | IIIA | _ | DJ STENTING | 2.1 | 1.2/9600 | Good/3 days | Gas resolved | continue | Good/MI |
| 12 | Savithri | 49 | F | 9942 | II | _ | ANTIBIOTIC | 2.5 | 1.1/9200 | Good/ 2days | Gas resolved | continue | Good/MI |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 13 | Kathayee | 61 | F | 10141 | IV | 4 | PCD- RT<br>LT DJ STENT | 1.1 | 4.9/<br>13000 | Progression/2 days | Persistent gas | RT.NEPHR ECTOMY | Poor / invasive |
| 14 | Devi | 47 | F | 10964 | II | _ | DJ STENTING | 2.2 | 1.2/9000 | Good/3 days | Gas resolved | continue | Good/MI |
| 15 | Roselin | 63 | F | 11630 | IIIB | 2 | PCD+DJ STENT | 2.2 | 1.3/7700 | Good/4 days | Gas resolved | continue | Good/MI |
| 16 | Mangayarkarasi | 46 | F | 12018 | II | _ | ANTIBIOTIC | 2.6 | 0.9/8000 | Good/3 days | Gas resolved | continue | Good/MI |
| 17 | Mohan | 64 | M | 12999 | II | _ | DJ STENTING | 2.5 | 1.2/7900 | Good/ 3 days | Gas resolved | continue | Good/MI |
| 18 | Powlin | 45 | F | 13567 | II | _ | DJ STENTING | 1.9 | 1.1/8700 | Good/ 3 days | Gas resolved | continue | Good/MI |
| 19 | Suseela devi | 65 | F | 14748 | IV | 4 | PCD-LT<br>RT DJ STENT | 1.0 | 4.5/20,000 | Alt.sensorium /2 days | Persistent gas | LT.NEPHR ECTOMY | Poor/ invasive |
| 20 | Kanagaselvi | 63 | F | 14835 | IIIB | 3 | PCD+DJ | 1.1 | 3.2/14000 | Shock +/2 days | Mass + | Open drainage | Good / invasive |
| 21 | Rajashekar | 47 | M | 15117 | II | _ | DJ STENTING | 2.6 | 1.0/8300 | Good/2 days | Gas resolved | continue | Good/MI |
| 22 | Kuppusamy | 48 | M | 15256 | II | _ | DJ STENTING | 2.5 | 1.0/9000 | Good/2 days | Gas resolved | continue | Good/MI |
| 23 | Raja Manikkam | 62 | M | 1076 | IV | 3 | B/LPCD+DJ STENT | 1.2 | 1.4/10000 | No mass/14 days | Obs.& gas resolved | continue | Good/MI |
| 24 | Muthupandi | 55 | M | 2089 | IIIA | 1 | DJ STENTING | 1.9 | 1.4/9100 | Good/5 days | Gas resolved | continue | Good/MI |
| 25 | Pandiselvan | 51 | M | 2951 | II | _ | ANTIBIOTIC | 2.8 | 1.1/7800 | Good/5 days | Obs. & gas resolved | continue | Good/MI |
| 26 | Rajeshwari | 59 | F | 3398 | IIIA | 1 | PCD+DJ STENT | 2.0 | 1.3/9850 | Good/7 days | No HUN & gas resolved | continue | Good/MI |
| 27 | Alagu Meenatchi | 55 | F | 4136 | II | _ | DJ STENTING | 2.6 | 1.0/7700 | Good/3 days | Gas resolved & no HUN | continue | Good/MI |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 28 | Karuppayee | 44 | F | 5839 | II | _ | DJ STENTING | 2.4 | 0.9/7900 | Good/2 days | Gas resolved/ No HUN | continue | Good/MI |
| 29 | Rakkammal | 66 | F | 6130 | II | _ | DJ STENTING | 3.0 | 1.2/7200 | Good/3 days | Gas resolved & no HUN | continue | Good/MI |
| 30 | Selvi | 43 | F | 6956 | II | _ | DJ STENTING | 2.2 | 1.1/6700 | Good/2 days | Gas resolved | continue | Good/MI |
| 31 | Sundharambal | 56 | F | 7177 | IIIB | 1 | PCD+DJ STENT | 2.3 | 1.3/9867 | Good/ 8 days | HUN & gas resolved | continue | Good/MI |
| 32 | Soundharam | 54 | F | 8570 | II | _ | DJ STENTING | 2.9 | 1.1/9800 | Good/ 4 days | Gas resolved | continue | Good/MI |
| 33 | Lakshmi | 57 | F | 9003 | IIIA | 2 | DJ STENTING | 1.9 | 1.4/9900 | No mass/5 days | Obs. & gas resolved | continue | Good/MI |
| 34 | Poochi | 53 | F | 10881 | II | _ | DJ STENTING | 2.8 | 1.0/7800 | Good/2 days | Gas resolved | continue | Good/MI |
| 35 | Kaaliamal | 59 | F | 10993 | IIIB | 2 | PCD+DJ STENT | 2.0 | 1.4/9000 | No mass/ 5 days | Obs. & gas resolved | continue | Good/MI |
| 36 | Chellamal | 51 | F | 11490 | II | _ | DJ STENTING | 2.2 | 1.0/8600 | Good/2 days | Gas resolved | continue | Good/MI |
| 37 | Muthammal | 65 | F | 12734 | IIIA | 2 | DJ STENTING | 1.9 | 1.5/10560 | Good/ 7 days | Obs. & gas resolved | continue | Good/MI |
| 38 | Saratha | 64 | F | 13561 | II | _ | DJ STENTING | 2.6 | 1.0/8600 | Good/3 days | Gas resolved | continue | Good/MI |
| 39 | Magalam | 68 | F | 14587 | IIIA | 2 | DJ STENTING | 2.5 | 1.4/9700 | Good/4 days | Gas resolved | continue | Good/MI |
| 40 | Parvathi | 67 | F | 14920 | IIIA | 3 | DJ STENTING | 2.2 | 1.4/10000 | Good /4 days | Obs.& gas resolved | continue | Good/MI |

**A PROSPECTIVE STUDY OF ROLE OF MINIMALLY INVASIVE APPROACHES FOR RENAL SALVAGIBILITY IN MANAGEMENT OF EMPHYSEMATOUS PYELONEPHRITIS**

*Dissertation submitted in partial fulfillment of the requirements of*

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*BRANCH IV – UROLOGY*

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This is to certify that this dissertation entitled "**A Prospective Study of Role of minimally invasive approaches for renal salvagibility in management of**